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SOME DANGER SIGNS IN CHILDHOOD.[1]

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WHEN I began work on this paper, I had in mind those major danger signs of which we think first in children—convulsions, pain, fever and vomiting; but on considering the matter more clearly, I realized that it is not these big things that are the most important danger signs, because they are so impressive that they cannot be neglected.

Think, for instance, of convulsions. We have all at some time or another been summoned frantically to the bedside of a child in convulsions. Usually when we get there it is all over; the child is lying quietly in a mustard bath, fæces and vomit are everywhere, and the household is excitedly racing about trying to think what else to do. And in nine cases out of ten the child recovers without turning a hair. There are exceptions, of course; the attack of convulsions may be the initial symptom of pneumonia or, worse still, of an encephalitis, or it may be the first fit of a true epilepsy, while a few years ago in Brisbane the possibility of lead encephalopathy could never be forgotten. But still, the average convulsion, a dietetic manifestation in an unstable child, is fraught with more terror to the parents than is any other condition of childhood, and with less reason.

Fever, too, may be very alarming, with little organic disease to show for it. The nervous child, it is true, is more prone to high temperatures with no discernible cause at all than is the placid child. I can remember the son of a colleague who had for years had bouts of fever with temperatures up to

[1] Read at a meeting of the Queensland Branch of the British Medical Association on October 1, 1937.

39·6° or 40° C. (103° or 104° F.), in whom nothing whatever could be found to account for the disturbance. I was the last of a number of clinicians who had unsuccessfully tried to solve the mystery. The attacks gradually became less frequent as he grew up, and, I think, ceased when he reached the age of about fifteen years.

One very common cause of symptomless infantile fever, too often missed, is *otitis media*, which is often singularly free from pain, although both membranes may be red and bulging.

The most important danger signs in childhood are often very slight, even transitory, but they are there to be looked for. The patient will seldom mention them; the parents will mention them even less. You will not find them if you adopt mass production methods in handling children, running them through your rooms like sheep through a crush; and I suppose that there is not one of us who cannot recall a bad instance of missing a vital sign because he did not look for it. In that 2 to 3 a.m. review of the past that even the most successful of us holds every now and then, the prominent figures are the sins of omission. They stalk past, not all to every one of us, but nobody can escape at least one of the batch: neither the child with intussusception that was not undressed, nor the transient squint that shouted for an ophthalmoscopic recognition of lead retinopathy, nor the child with pneumonia that underwent a laparotomy because the chest was not examined, nor those other mistakes we can all make, even the youngest of us.

So to-night I shall speak of a number of small signs, which, although apparently insignificant, are the key to diagnosis in conditions not at all trivial.

First, consider what is to many people the most obvious thing about a baby—its cry; some will tell you that a baby always cries, and that little notice need be taken of it. But if there is one thing more certain than another, it is that even a small baby has reasons and prejudices, likes and dislikes; and his only way of expressing these feelings is by crying. It is also probable that the first year of a baby's life is more unhappy than any of the following sixty-nine, so that even the most stoical of babies may complain under such conditions.

My old friend and teacher, the late Dr. Jefferies Wood, told us once that there are four basal cries in infancy: hunger, pain, anger, and boredom. No doubt many people will agree with the first three, but will question the last. It is a cry difficult to describe, but it may be always heard at railway stations on holiday evenings, and it is too often heard in hospitals and baby homes. For far too frequently is it forgotten that a baby is not a piece of machinery, to be wound up, cleaned, refuelled, picked up and set down at certain more or less regular intervals; but a breathing, living and thinking individual. A baby thinks differently from an adult, but he does think, and we ought to make his already very difficult entry into our society as easy as possible.

Take, for instance, the simple matter of going to bed. In too many cases baby's last hour of the day is a time of interest and brightness, incident and enjoyment, when suddenly, for no reason at all, this pleasant party stops. He is dumped down, the friendly light goes out; worse still, those delightful people change into cruel tyrants; they go away, leave baby unprotected against the darkness and loneliness, or if they do return in response to his piteous cries for help, it is with hard angry looks and even blows. What is baby to think? Either that they are really at heart cruel and callous, or else that he has been terribly wicked to be treated like this. Neither state is conducive to sleep, so baby cries and stays awake until, utterly exhausted by grief and exertion, he falls asleep. Too often the psychical damage has been done, and I think I am right in calling that cry of weariness and unhappiness a danger sign.

Take an altogether different cry in a sick child—a sudden, piercing scream, followed by a long interval of silence, and the same thing again, the child relapsing into stupor between each cry. Our medical forefathers named it the meningeal cry, and when it is present (for it is not a constant feature) it is as diagnostic as Kernig's sign.

Another cry which will make a diagnosis for us is the scream of the enraged or frightened mentally defective; there is something in it which falls on the ear of experience and sets the brain planning ways in which the horrible truth can be broken to the child's parents.

But of all the cries, the most miserable is that of the child with advanced pink disease; here there are exhaustion, pain, hopelessness and resentment.

There is another most significant cry in children under a year old. An infant, usually a healthy, breast-fed boy, wakes suddenly from sleep with a cry of pain. His face is white and drawn; he wriggles with pain and perhaps vomits. Soon the pain passes off; he falls asleep, only to wake again with the same cry and the same drawn look. Perhaps this time he doubles up his legs on his abdomen; his bowels act, and a normal stool with some blood-stained mucous jelly is passed.

This process is repeated, and at last the family realizes that something is wrong and takes him to a doctor, who, let us hope, will not accept the family diagnosis of "gasteritis", but will recognize the true gravity of intussusception. And, paradoxically, the absence of a cry may be a danger sign of the first importance; I always feel uneasy when a proud mother tells me of any child, infant, toddler, or child of early school age: "He never cries; he is always so good and placid." Yes, and so is a cabbage; but children are normal animals, gradually and with many pains settling themselves into our social life, and it is not fair to expect them to do so without some reaction. The child under one year of age who never cries, is always placid, sleeps to order and is content "to be fed, washed, dressed and changed" may be a normal child; but he may be underfed, mentally defective or over-drilled. None

of these is a desirable state, and none should be forgotten.

In older children a sudden appearance of goodness, docility and placidity may really hide a mental crisis of no small importance.

It does not seem at first that there are danger signs in learning to walk, but usually late walking may be caused by one of three conditions: cerebral defect, muscular dystrophy, or congenital dislocation of the hip. I have lumped spastic paralysis, mental defect and cretinoid idiocy together as cerebral defect. The mongol, the spastic diplegic and the cretin alike cannot use their limbs because the controlling power is absent, while in the case of the cretin and the diplegic, early recognition and treatment may make all the difference between helpless invalidism and an active, almost normal life. On the physical side, it is most important to recognize muscular dystrophies early; some of these respond to treatment surprisingly well.

This is not all; the birth of one dystrophic child may be a warning of more to come, and the parents should at least be told of the likelihood of this. If they choose to run the risk of a dystrophic family, that is their affair; but the doctor's duty will have been done if he warns them.

The recognition of congenital dislocation of the hip at an early stage means a normal limb; failure means a lifetime of deformity, and, in the case of a woman, danger to life in every confinement.

Not merely his gait, but the way a child stands, may be of importance at a very early stage. The best example of that is the "ptotic stance", which is easily recognized when it is well developed; but by that time is much more difficult to treat. In this condition, the child (boy or girl, though usually a girl) stands with shoulders drawn back, abdomen pushed forward and weight mostly on the heels. Often, too, there are flat foot and knock-knee as well. Usually these children are alert and bright, very sensitive and nervous. They are unduly successful at school, and studious rather than athletic. They are over height and under weight; their appetite is capricious, and they are especially prone to like foods that "are not good for them", and to dislike the articles so strongly recommended by the League of Nations. Often they are constipated, and this is a further cause for anxiety to their parents, who treat the constipation with various forms of aperients, especially roughage.

The danger is in the future more than the present; they are apt to pass into that deplorable condition described by Robert Hutchison as "the chronic abdomen". Whether this is an organic or a functional disease I will not argue here; but there is no doubt of its existence. These children respond well to treatment of the right sort, and, as people of superior intelligence, are well worth the effort necessary to save them from later chronic illness. One of the most deplorable examples is a woman who comes to see me at times, who has had her appendix out (of course!); her right kidney out; her gall-bladder out; her colon "pexed"; all sorts of things done in her pelvis; her antra radically treated; her septum resected; her tonsils taken out; and eight or nine mastoid operations. But she is an extreme case.

The next sign of very great importance to which I shall draw your attention is the child's expression; and a child's expression may be significant within a few hours of its birth. The mongol or cretin, for example, may be recognized at the age of a few days old, though, mercifully, the mongol may not be guessed at for some years. In later years, of course, the face in each of these conditions is characteristic. It is curious how often it is the first impression which matters in these cases. It is easy to see a child passing through the out-patients' department and to say, "Mongol"; yet, when a detailed examination is made of the same child, the signs may be slight and conflicting. But that first impression was right, and as a mongol the child grows up.

In the diagnosis of disease in later childhood, that flash of recognition is often invaluable. In the toxic stage of severe diarrhœa the child assumes an expression difficult to describe, but very characteristic; too often it means death within twenty-four hours. The marasmic baby, wasted but not toxic, has quite a different expression, while the child starved from pyloric stenosis acquires a very striking frown, and a hungry, anxious look.

The syphilitic facies is becoming a rarity, while the child with scurvy or rickets is a museum specimen.

The face in pink disease is characteristic, and the diagnosis is generally obvious.

It is seldom that we see a *risus sardonicus* sufficiently early in tetanus for it to be a special danger sign; when it is present it seems to emphasize the severity of the infection.

The pneumonia face, with its flushed cheeks, anxious expression, bright eyes, and twitching nostrils, and accompanied by the patient's grunting respirations, may for days be the only recognizable feature of the disease, and may be the final point which settles whether the child has pneumonia or an acute appendicitis.

Another face, which is fortunately becoming steadily rarer nowadays, is that of the chronic juvenile nephritic: the pale face, the heavy lines on forehead and cheeks, and the indefinable something that makes the deplorable diagnosis complete. Nor are these young people by any means all weaklings; one young patient, suffering from nephritis due to lead poisoning, was, despite paralytic flat feet, a footballer, swimmer and wrestler, in hard training till his death at nineteen years of age from uræmia. But his face was characteristic.

While we are on the subject of pale faces it is important to remember that while pallor does not mean anæmia, anæmia does mean pallor. Now, anæmia in childhood may be merely nutritional; it may be due to lead poisoning, or it may be due

to some severe blood disease like leuchæmia. In any case, pallor calls for investigation, and there is only one yardstick for pallor, the hæmoglobinometer; and there is only one yardstick for anæmia, a complete blood examination. It is disconcerting, to say the least, to have the child, whose parents have been assured that it had just a little anæmia, go on to a fatal leuchæmia; but this can happen, if full investigation is not made.

Eczema makes its first appearance on the face, usually on the cheeks—a little, scaly, irritable area, with a slightly weeping skin when the scales are rubbed off. Whether we believe that eczema is a simple allergy, as some cases suggest, or a fat dyscrasia, as others seem to indicate, there is no doubt of the ease of its treatment in the early stages and of the extreme difficulty of treatment later. And eczema is a dangerous disease, as any disease must be which confines a child in hospital or in dressings and bandages for months on end; and the rapidity with which a simple facial eczema can spread and involve other areas of the body must be seen to be believed.

The facial appearance may be the outstanding feature, too, in that very grave chronic malady, bronchiectasis, though signs in the chest may be almost absent.

I now turn to a group of danger signs associated with pain, and at the beginning I would emphasize the importance of very minor degrees of pain. Pain sufficient to make a child complain is always serious, until we have proved by examination that it can be neglected.

Yet, at the same time, it is surprising how stoical children may be when there is apparently a very considerable degree of suffering, and how very great may be the difference between one child and another in this respect. It is merely another example of individual difference. Unfortunately, children sometimes have another reason for refusing to admit the existence of pain, and that is fear of the treatment it may entail.

Of all the pains from which small infants suffer, the one which arouses most controversy is that produced or not produced by the eruption of the first teeth. On one hand we have such shrewd observers as Dr. Jefferis Turner, of this city, or Feer, of Zurich, who stoutly affirm that teething never produced anything except teeth; on the other hand, we have such acute clinicians as G. F. Still or John Thomson, who admitted that certain upsets, such as pain in the gums, earache, digestive disturbance, or even bronchitis, might be induced by the eruption of the milk teeth. I myself incline to the latter view. We must remember that the eruption of a tooth is a most complicated matter, with three factors at least involved: the absorption of the gum above the tooth, the push upward of the tooth itself, and the following movement of the permanent tooth behind. If for any reason these do not completely synchronize, there seems reason to expect at least local pain and irritation.

Curiously enough, I am not aware that any of those who have conducted analyses into the very early years of life have mentioned teething as a source of physical pain; as a source of psychical pain there is ample evidence of its importance. The great danger of teething in infancy lies not in its neglect, but in the over-emphasis laid upon it; meningitis, *otitis media*, squint, intussusception, pneumonia, leuchæmia, are only a few, remembered at random, of the conditions diagnosed originally by parents (and too often, I am afraid, by overworked medical practitioners) as "teething". Moreover, of course, in every digestive upset teeth get the first blame.

I feel that only when we have thoroughly examined a child, and are convinced that there is neither *otitis media*, tonsillitis, pneumonia, incipient measles or scarlet fever, pyelitis, meningitis, osteomyelitis nor pink disease, then, and then only, may we recognize that the dribbling, the red and irritable gums and the disappearance of symptoms with eruption point to the condition being due to "teething".

Headache must be divided into headache with fever and headache without fever.

In the first group come such diseases as dengue, influenza, and half the acute febrile diseases of childhood; but meningitis, especially influenzal meningitis, is too often overlooked till the condition is obvious, and the possibility of treatment is even more hopeless than at the beginning.

At the present time the combination of headache, fever and a stiff back should be enough to alarm any doctor, especially if he has read Dr. Mostyn Powell's admirable article in THE MEDICAL JOURNAL OF AUSTRALIA of September 11, 1937.

Afebrile headache is less common in children than in adults. It should suggest one of at least three very serious conditions: eye strain, lead encephalopathy or cerebral tumour. Every headache of this class should at least lead to an ophthalmoscopic examination. Lead encephalopathy is a rare disease nowadays; this is a greater reason for remembering its possible presence.

Headache is apparently not nearly so constant a feature of cerebral tumour in children as it is in adults. Vomiting, often occurring in the early morning and not associated with food, is generally a much earlier sign. Even in a child with all the signs of advanced cerebral pressure, headache may be quite insignificant; the child may, in fact, be simply drowsy and depressed.

We are fortunate in this country that tuberculous meningitis is a rarity. Where the disease is prevalent, its onset may be marked merely by headache and by a temperature a little above normal at first; otherwise there may be little to show. Transient squint, however, is here very common, the squint becoming fixed later on. One of the few patients with this condition I have seen here was admitted to the Children's Hospital with a diagnosis of plumbism, and an optimistic prognosis was

given. Not sufficient importance was attached to the temperature, and only when the child's condition grew steadily worse instead of better following repeated lumbar puncture, did a revision of the diagnosis become inevitable.

Of all the serious diseases which may attack children after two years, three may have pain in the limbs as their only manifestation. These are lead poisoning, rheumatism and osteomyelitis. The first is growing more and more uncommon, but odd cases occur every now and then; it is too often diagnosed as rheumatism and allowed to go untreated. The patient is afebrile, looks ill and pale, and has some source of lead at hand; usually abdominal pain is also present. The pains in the limbs are more often cramping than not, and are often in the neighbourhood of, rather than in, joints; for example, they occur among the hamstring tendons behind the knee.

Rheumatism is common enough in children in this country after the second birthday. Generally there are slight fever and signs of throat infection, but sometimes the child may show little except a disinclination to play, and a slight limp on walking. The heart, however, will often be found to have suffered serious changes, and the blood sedimentation rate is more rapid than normal. Children with gross cardiac changes often come into hospital of whose previous illness nothing more can be remembered than "growing pains".

Osteomyelitis and rheumatism are frequently confused; if all examples were straightforward, textbook cases, there would be little difficulty; but we have all seen the atypical forms, and we can all remember our difficulty and worry. One useful aid is that in rheumatism rigors are very unusual; in osteomyelitis they are quite common.

In this country tuberculosis of the hip is, I am glad to say, a rare disease; when it does occur it is often missed, and all for lack of interest in an insignificant limp and pain in the knee.

Abdominal pain is the best recognized of all danger signs. Few will fail to examine the abdomen of any child who complains of pain, and in childhood the local signs are generally very direct and straightforward. There is, of course, an important exception: alveolar (lobar) pneumonia. In this condition the signs may mimic an "acute abdomen", and though usually the inspiratory grunt, the dyspnœa, and the tendency to delirium are more common in pneumonia than in general peritonitis, even the shrewdest clinician may be tricked. Radiography may be most helpful, but the best guide is a careful clinical examination, with due attention to nostrils, breath sounds and local rigidity.

The pain of lead poisoning is peculiar. It may come on at any hour of the day or night; it bears no relation to food; there is no rise of temperature; it is situated in the epigastrium rather than in the lower part of the abdomen, and, most characteristic of all, it is relieved by pressure. Practically all other abdominal pains in childhood are increased by pressure. Lead poisoning is much less common nowadays, but a few years ago it held in Queensland as a cause of abdominal pain the position which spinal caries holds in the South; it had always to be kept in mind when an examination was made until it could be finally excluded.

For our medical grandfathers, intestinal worms shared pride of place with teething as the most common cause of juvenile sickness. Today we are not so sure; but I believe that colicky pain of an indefinite nature, restlessness at night, loss of appetite and *pruritus ani* may be caused by threadworms. This condition is often confused with juvenile dyspepsia, and the child is dosed with santonin when he really needs a dietary. The diagnosis of threadworms in the appendix is made, I think, more often than is justified, although there is no doubt that in this position they do give rise to mild attacks of appendicitis; but I have seen this diagnosis made in one case of cyclic vomiting, and the condition once given as a cause of "dilated heart".

Respiratory obstruction is so grave a condition that it naturally calls for early attention, and it may be present from birth. Of this there are two important causes: laryngeal malformation and persistent enlarged thymus. Diagnosis of the latter made after X ray examination is very misleading; there is only one sound method of diagnosis: laryngeal examination (if necessary, under anæsthesia) by an expert. If the condition is not dealt with, the result is pitiful: a stunted, distorted child, always a prey to lung infections and finally dying of them.

In older children sudden laryngeal obstruction must always rouse the suspicion of a foreign body. The obstruction of spasmodic laryngitis, of measles, or of laryngeal diphtheria is a gradual process.

Finally, there are two danger signs which are often disregarded till too late, when permanent damage has been done: enuresis and night terrors. Parents and doctors alike are apt to treat these distressing conditions with a curious optimism, and to say: "Oh, he'll grow out of them."

Well, he may or may not. I have seen bedwetters twenty-five years old who were social outcasts, and the final state of at least one child who had suffered constantly from night terrors is not a pretty one. In any case, the mental pain and humiliation these small sufferers undergo call for alleviation as much as does toothache. Both conditions may have a physical cause; both are more commonly nervous in origin.

Malformation of the urinary tract is one very important, though not very common, cause of enuresis. It is more likely to be present when there is day and night incontinence, and its detection generally requires a urogram. The condition is one of extreme dilatation of the ureters and hydronephrosis, generally with no blocking anywhere in the urinary tract. It generally terminates fatally as a result of kidney failure. It is curious how

apparently well and healthy such children may be at quite a late stage of the disease.

Adenoid obstruction may often keep up enuresis; removal of adenoids may effect great improvement. Otherwise enuresis is generally the sign of severe mental conflict, and is best treated by a complete temporary change in environment. The most successful drugs in the treatment are thyreoid extract, phenobarbital and belladonna, the last up to doses large enough to produce central disturbance (which is probably how the drug acts, rather than by any action on the bladder nerves of the sacral plexus). Of special importance is a sudden onset or increase in the habit; it nearly always means an added emotional strain. One small girl, till she was twelve, always had enuresis after a quarrel between her father and her mother, but at no other time.

Night terrors, it is true, are at times caused by respiratory obstruction, and are cleared up by removal of tonsils and adenoids. (But we must never forget the psychical effect of this operation on a sensitive child.) There are other night terrors, however, which can be explained only after an investigation of home surroundings, of school troubles, of the attitude to parents, to dogs and to various "dangers". Here, again, a complete change, *plus* a small amount of sedative, is the most satisfactory form of treatment, unless the cause is easily discernible and removable.

In going over this paper I have been struck by two points: first, the emphasis on the obvious, and, secondly, the prominence given to the nervous factors.

As to the first, another of my teachers, the late Dr. A. V. M. Anderson, used to say: "The most common fault in medicine is an inability to appreciate the obvious." Moreover, my experience at the Hospital for Sick Children is that these are the sort of things which are being missed.

As to the second, children's ailments to our medical grandfathers mostly meant worms and teething; to our fathers, they meant summer diarrhœa and syphilis; to this generation, they mean nutritional and nervous disorders.

The work of teachers, welfare workers, psychiatrists and child guidance workers is slowly giving us new and changing conceptions of the mental processes of the child, revealing, too, the importance of those mental processes in a child's physical and mental life. Much of this work is not yet applicable to the ordinary child patient; but it is surprising that many a medical man, who would be upset were his knowledge of the latest views on B_x to be questioned, is content to rub along with the child psychology of last century.

But again I warn you that these ideas are of little practical importance in the sheep-trucking methods of our present out-patient departments. They can be, and are being, successfully used by many practitioners in the patient's own family circle.

I can only hope that what I have said will provoke those who disagree with me to stand up and say so.

A DOSAGE SYSTEM OF CERVICAL CANCER.

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CANCER of the cervix is insidious in onset and unaccompanied by pain, for which reason few early cases are seen. The extent of the growth is impossible to estimate. In many cases there appears to be extensive infiltration of the broad ligaments, which disappears after rest in bed, and is therefore obviously inflammatory and not malignant in character. On the other hand, at operation or *post mortem* the extent of the disease is frequently found to be much more extensive than any clinical examination can reveal.

Surgical treatment under the best conditions does not yield quite such goods results as radiation methods, and is associated with a large operative mortality and morbidity.(1) For this reason this paper is confined to radiation methods, and in particular those in which γ rays are used.

The radiation dose which will cause death or retrogression of cells is an interesting question. Indices of malignancy have been prepared.(2) Though we all recognize that broadly the carcinomata in which there are large active cells and little intercellular tissue are in general more radiosensitive than those with both well-marked alveolar structure and intercellular tissue, often two apparently similar cases will react in an entirely different manner. In one sloughing and much reaction will occur, while in the other healing will take place with little reaction. Sometimes, although rarely, irradiation seems to have little effect on a growth which would appear to be of a radio-sensitive type. Apart from anæmia and other conditions which will profoundly influence the effect, it may be found that entirely different results are produced in otherwise apparently healthy women by exactly the same dose given under apparently the same conditions.

Apart from the above considerations, difficulties are introduced in delivering an adequate dose of radiation to the area to be treated without damage to surrounding structures. The *cervix uteri*, the upper part of the vagina, and the lower part of the body of the uterus form a cylinder of tissue which can be removed surgically or treated by irradiation with X rays or γ rays. As it is impossible to determine the exact extent of the growth, the whole cylinder should be evenly irradiated. The mucosa of the fundus must be entirely destroyed or its secretions may cause a hæmometra or pyometra after the cervix has been destroyed.

The cylinder of tissue to be treated is surrounded by the bladder in front, the ureters at each side, and

the rectum behind. The ureters and bladder are not very radio-sensitive; but the rectum is highly radio-sensitive. This distribution renders the use of X rays difficult.

The cylinder of tissue treated may itself vary in dimensions very widely, and a fixed dosage system suitable for one case may give unsatisfactory results in another. An examination of the literature dealing with methods of treatment by radium indicates, however, that various fixed dosage systems are usually employed.[3][4][5][6][7] A flexible dosage system, adjustable to the varying geometrical requirements of individual cases, is difficult to realize with radium containers of fixed sizes and strengths. In the method described here this objection is overcome by the use of radon.

Geometrical Considerations Involved.

The object has been to develop a dosage system covering the range of sizes of *cervix uteri* seen in practice and giving uniform known doses both to various points in the treated tissues and to adjacent structures.

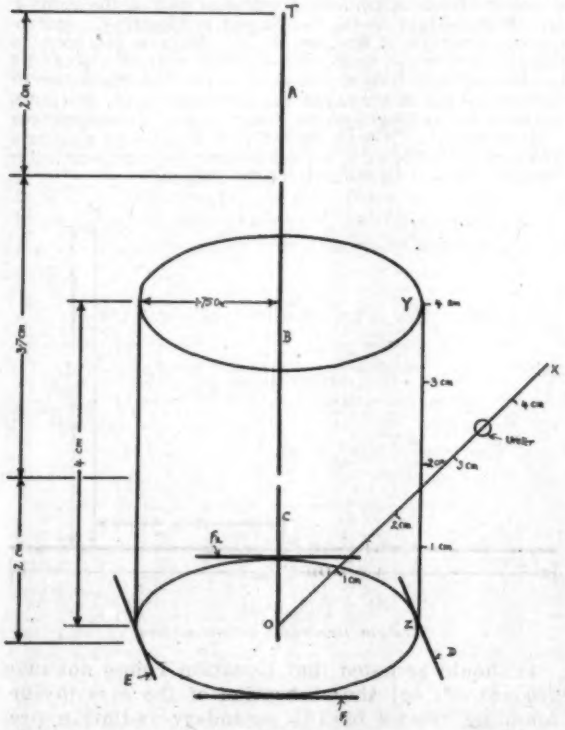

FIGURE I.
Typical arrangement of active lengths with respect to cylindrical region treated.

For the purposes of calculation, a definite cylindrical volume of tissue has been chosen. This cylinder is illustrated in Figure I, and is of a diameter of 3·5 centimetres and height 4·0 centimetres, with its axis *OT* along the centre of the uterine cavity and its base at the cervix. Actually the volume of tissue treated (Figure II) consists of the above cylinder extended downwards for a distance of about 1·0 centimetre; that is, it comprises the upper end of the vagina, the cervix, the parametria, and that portion of the body of the uterus that extends from the junction of the body and the cervix to the point of most distant treatment, *Y*. The latter point has been arbitrarily chosen.

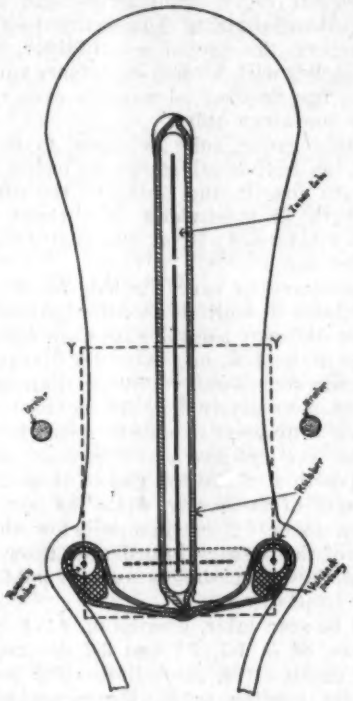

FIGURE II.
Sectional diagram, showing tubes and pessary in position for a typical case.

The variable geometrical factors which have been taken into account are as follows:

1. Variation of the length of an inner radon container to suit uterine cavities of lengths between 4·5 and 8·5 centimetres. Cases have been considered for five lengths covering this range.
2. Variation of the linear strength of the inner radon container along its length.
3. Use of a square of four tubes or a circular tube around the cervix.
4. Use of squares of different sizes to allow for variations in the size of the cervix. Three cases have been considered in which squares of sides 3·5, 4·0 and 4·5 centimetres in length respectively have been used.
5. Position of square pessary relative to the cervico-vaginal junction. Two cases have been considered in this respect.

Thus thirty different types of cases have been developed. In addition, the dose received by the ureters has been calculated and reduced to a relatively low value for each of the cases considered. For the purposes of calculation a ureter has been assumed to be situated at a distance of 3·5 centimetres along a line *OX* at 45° to the axis of the

cylinder running through the centre *O* of the base (see Figure I). Also the most radio-sensitive structure adjacent to the area treated is the rectum, which is protected by means of a lead mantle in the special pessary described below.

The Arrangement of Radon Tubes.

For the attainment of a high treatment dose in the cylindrical region considered (see Figures I and II) while minimizing the relative dose delivered to the ureters, the use of a container inside the uterus, together with several containers surrounding the cervix, has decided advantages over the use of the inside container only.

The inside radon tube is made to fit the full length of the individual cavity in order to supply an adequate dose to the lining of the uterus. Its active length is subdivided into three portions: *A*, at the inside end; *B*, at the centre; and *C*, at the cervical end.

It is necessary to vary the lengths of *A* and *B*, and sometimes to omit *A*, to suit the length of the cavity, and different lengths have been distinguished as A_1, A_2, B_1, and so on. Also the linear strength of *B* is made considerably greater than that of the other component active lengths in order to obtain the desired difference in dosage rates between the ureter and the cylinder. It may be noted here that the position of the top end of *A* is determined by the length of the cavity, while the bottom end of *C* is taken to be 0·2 centimetre below the level of the plane of the pessary in order to allow for slight bulging of the rubber bands used to hold the inner container in position.

As will be seen later, lengths of *A*, of 1·0 and 2·0 centimetres, of *B*, 1·7, 2·7 and 3·7 centimetres, and of *C*, 2·0 centimetres, are suitable for building up total active lengths to fit the range of sizes of cavity occurring. These component lengths are specified as follows:

Length A_1.—Active length, 2·0 centimetres; centre, 6·5 centimetres above the base of the cylinder in Figure I.

Length A_2.—Active length, 1·0 centimetre; centre, 6·0 centimetres above the base of the cylinder in Figure I.

Length B_1.—Active length, 3·7 centimetres; centre, 3·65 centimetres above the base of the cylinder in Figure I.

Length B_2.—Active length, 2·7 centimetres; centre, 3·15 centimetres above the base of the cylinder in Figure I.

Length B_3.—Active length, 1·7 centimetres; centre, 2·65 centimetres above the base of the cylinder in Figure I.

Length C.—Active length, 2·0 centimetres; centre, 0·8 centimetre above the base of the cylinder in Figure I.

Four separate radon tubes, in all cases equal in strength to each other, and with active lengths of 2·0 centimetres, are used in the form of a square pessary to surround the cervix. In a previous paper[8] dealing with portion of the calculations involved herein, it has been shown that the use of a square pessary instead of a circular uniform source makes little difference as far as relative doses are concerned; the square is, of course, more easily constructed for practical application.

Square pessaries with sides of three different lengths are used to cover variations in size of the cervix. The plane of the pessary is normally that of the base of the cylinder shown in Figure I. This ideal position is not, however, always reached in practice, since abnormal shape of the fornices sometimes causes the pessary to be displaced about 1·0 centimetre downwards. Both types of cases have been considered in the calculations. The normal ones are specified as Cases I, II and III, and the abnormal ones as I′, II′ and III′. In Figure I the four tubes under consideration are indicated as *D*, *E*, F_1 and F_2.

The Method of Calculation.

Since the component active lengths of the radon tubes used are straight line radio-active sources, one formula[9][10][11] suffices for the approximate calculation of the relative doses delivered at any chosen point. The individual effects may then be added together. The formula used is as follows:

Equation I:

$$R = \frac{mN}{b}\left[\tan^{-1}\left(\frac{a+L/2}{b}\right) - \tan^{-1}\left(\frac{a-L/2}{b}\right)\right] \text{ Röntgen per hr.}$$

where *R* (Röntgen per hour) = dosage rate at the point *P* (see Figure III); *m* (millicurie per centimetre) = instantaneous strength of the source; *N* (Röntgen per hour) = number of Röntgen units delivered per hour at a point 1·0 centimetre away from a point source of 1·0 milligramme of radium, taking into account the screenage used; *L* (centimetre) = active length of the linear source; *a* (centimetre) = displacement of *P* from the centre O of the source along a line parallel to the axis; *b* (centimetre) = the perpendicular distance from *P* to the axis of the source.

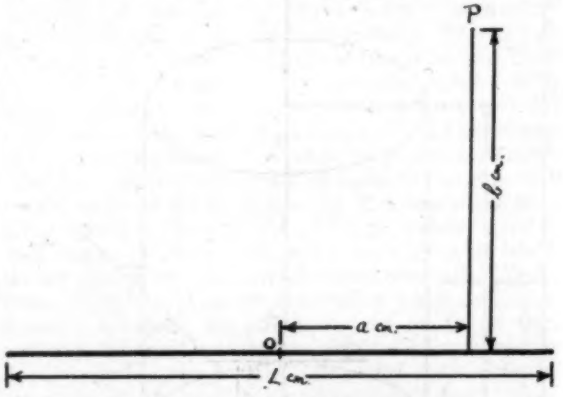

FIGURE III.
Straight line radio-active source.

It should be noted that Equation I does not take account of: (*a*) the absorption of the rays in surrounding tissues or (*b*) secondary radiation produced in these tissues. These effects are usually neglected,[9][11] as sufficiently reliable data are lacking, and as the effects are small and to some extent will compensate each other.[7]

Consideration of Figure I shows that the dose is a minimum for all points in the assumed cylindrical region at the point marked *Y* on the edge of the top end of the cylinder. For comparison, dosage rates have been calculated for points (x = 1·0, 2·0, 3·0 and 4·0 centimetres) along the line *OX* at 45°

to the axis *OT* running through the centre *O* of the cylinder base, and for points (y = 1·0, 2·0, 3·0 and 4·0 centimetres) along a line *ZY* parallel to and 1·75 centimetres away from the axis of the cylinder, lying in the same plane as *OT* and *OX*.

Under these conditions a ureter is at a point x = 3·5 centimetres, and the point *Y* in the cylindrical volume receiving the minimum dose is y = 4·0 centimetres.

Values of the factor *K*, where $K = \frac{R}{Nm}$ (Equation II) have been found for each x or y point considered by substitution in Equation I, the required values of a and b being determined easily from the geometry of the system.

In Table I are enumerated the calculated values of *K* for a series of points near four tubes, *D*, *E*, F_1 and F_2, arranged in squares with sides either 3·5, 4·0 or 4·5 centimetres long; in all cases each of these tubes has an active length of 2·0 centimetres, which is situated symmetrically with respect to the centre of a side of the square. The total value of *K* is also given for each of the three squares.

Various arrangements of active length along the axis *OT* of the uterine cavity have also been considered; as has already been indicated, three active lengths, *A*, *B* and *C*, are used in actual practice. Values of the factor *K* for these active lengths are set out in Table II for the same series of x and y points as were considered in Table I; but whereas in Table I the resultant effect for a square is given by the sum of the values of *K* for four tubes (*D*, *E*, F_1 and F_2), since the four tubes are assumed to be equal in linear strength m, in Table II the lengths *A*, *B* and *C* have different strengths and must therefore be considered separately.

The Choice of Arrangement of Active Lengths.

Three factors limit the arrangement of the active lengths:

1. The four containers around the cervix should be equal in length and strength in order to avoid variations in the amount of radiation reaching any given point in the event of possible rotation of the pessary, and to maintain equal dosage rates at points situated symmetrically around the axis of the cylindrical volume.

2. The square should be of suitable size to fit the upper part of the vagina.

3. The sum of the active lengths along the axis should approximate to the length of the uterine cavity, so that the inner lining of the latter may be given an adequate dose.

With these considerations in mind, cases have been considered combining various lengths of cavity and sizes of cervix. They are specified in Table III and cover the range of dimensions usually found in practice. In the first column a case number (I, II *et cetera*) is given. The second column gives the lengths of the uterus, and the third the lengths of a side of the square pessary. In the remaining columns are given the assumed linear strengths (m) of the various active lengths.

The case specified as I*a* covers the method of treatment employed before the introduction of this dosage system; Case I*b* is now used in its place.

TABLE I.

Values of K for a Series of Points near Four Active Lengths, each of 2·0 Centimetres, in a Square with Side of 3·5 Centimetres, 4·0 Centimetres, or 4·5 Centimetres.

Point Considered. (See Figure I.)	Square with Side of 3·5 Centimetres. Normal Position.					Square with Side of 4·0 Centimetres. Normal Position.					Square with Side of 4·5 Centimetres. Normal Position.				
	D.	E.	F_1.	F_2.	Total for 3·5 Centimetres Square.	D.	E.	F_1.	F_2.	Total for 4·0 Centimetres Square.	D.	E.	F_1.	F_2.	Total for 4·5 Centimetres Square.
x=1·0 cm.	0·293	1·064	0·470	0·470	2·30	0·244	0·811	0·384	0·384	1·82	0·209	0·625	0·317	0·317	1·47
x=2·0 cms.	0·162	0·834	0·282	0·282	1·56	0·143	0·758	0·248	0·248	1·40	0·127	0·669	0·220	0·220	1·24
x=3·0 cms.	0·101	0·371	0·169	0·169	0·810	0·092	0·414	0·155	0·155	0·816	0·084	0·414	0·143	0·143	0·784
x=4·0 cms.	0·068	0·211	0·106	0·106	0·491	0·063	0·222	0·101	0·101	0·487	0·059	0·231	0·096	0·096	0·482
y=1·0 cm.	0·148	1·568	0·288	0·288	2·29	0·131	1·49	0·254	0·254	2·13	0·115	1·30	0·221	0·221	1·86
y=2·0 cms.	0·122	0·464	0·197	0·197	0·980	0·108	0·453	0·181	0·181	0·923	0·098	0·440	0·164	0·164	0·866
y=3·0 cms.	0·092	0·214	0·132	0·132	0·570	0·085	0·212	0·124	0·124	0·545	0·078	0·209	0·115	0·115	0·517
y=4·0 cms.	0·070	0·122	0·089	0·089	0·370	0·066	0·122	0·087	0·087	0·362	0·061	0·120	0·082	0·082	0·355

TABLE II.

Values of K for Component Active Lengths along the Axis of the Cavity.

Point.	Inside End.		Central Portion.			Outside End.
	A_1.	A_2.	B_1.	B_2.	B_3.	C.
x=1·0 cm.	0·062	0·034	0·608	0·559	0·463	2·690
x=2·0 cms.	0·074	0·044	0·687	0·619	0·503	0·791
x=3·0 cms.	0·088	0·052	0·547	0·466	0·341	0·321
x=4·0 cms.	0·095	0·056	0·392	0·314	0·206	0·166
y=1·0 cms.	0·063	0·036	0·440	0·387	0·303	0·586
y=2·0 cms.	0·089	0·052	0·698	0·612	0·469	0·449
y=3·0 cms.	0·136	0·085	0·891	0·747	0·503	0·268
y=4·0 cms.	0·226	0·143	0·916	0·672	0·356	0·157

TABLE III.

Specification of Active Lengths for a Variety of Cases with Various Sizes of the Uterus.

Case Number.	Length of Uterus in Centimetres.	Length of Side of Square Pessary in Centimetres.	Linear Strengths of Component Active Lengths at Time of Insertion in Millicuries per Centimetre.			
			Inside Axial Part *A*.	Central Axial Part *B*.	Outer Axial Part *C*.	Square of Four Tubes.
Ia	8·0 or 8·5	3·5	A_1 .. 6·24	—	6·24	6·24
Ib	8·0 or 8·5	3·5	A_1 .. 2·50	B_1 .. 5·00	2·50	2·50
II	8·0 or 8·5	4·0	A_1 .. 2·50	B_1 .. 5·00	2·50	2·56
III	8·0 or 8·5	4·5	A_1 .. 2·50	B_1 .. 5·00	2·50	2·61
IV	7·0 or 7·5	3·5	A_2 .. 3·96	B_1 .. 5·00	2·50	2·50
V	7·0 or 7·5	4·0	A_2 .. 3·96	B_1 .. 5·00	2·50	2·56
VI	7·0 or 7·5	4·5	A_2 .. 3·96	B_1 .. 5·00	2·50	2·61
VII	6·0 or 6·5	3·5	—	B_1 .. 5·62	2·50	2·50
VIII	6·0 or 6·5	4·0	—	B_1 .. 5·62	2·50	2·56
IX	6·0 or 6·5	4·5	—	B_1 .. 5·62	2·50	2·61
X	5·0 or 5·5	3·5	—	B_2 .. 7·70	2·50	2·50
XI	5·0 or 5·5	4·0	—	B_2 .. 7·70	2·50	2·56
XII	5·0 or 5·5	4·5	—	B_2 .. 7·70	2·50	2·61
XIII	4·0 or 4·5	3·5	—	B_3 .. 17·2	0·68	0·68
XIV	4·0 or 4·5	4·0	—	B_3 .. 17·2	0·68	0·695
XV	4·0 or 4·5	4·5	—	B_3 .. 17·2	0·68	0·71

The Summation of Effects of Individual Active Lengths.

Equation II shows that the dosage rate at a point near a single active length is proportional to Km, since $\frac{R}{N} = Km$, and since N has the same value throughout if a constant screenage is employed. The sum of the effects of a number of lengths is given by the sum of the corresponding values of Km. Further, as was indicated under the heading "Method of Calculation", the values of K for the four tubes in the square may be added together, since m is the same for each of them.

Values of $\frac{R}{N}$, for the series of points 1·0, 2·0, 3·0 and 4·0 centimetres along the x and y lines (Figure I) have been calculated for each of the arrangements of active lengths (Ia to XV) specified in Table III; they are given in Table IV. In the bottom line of Table IV are included values of the ratio of the dosage rate at the ureter (that is, the point x = 3·5 centimetres) to that at the adjacent point y = 4·0 centimetres in the cylinder for each case considered. Consideration of the old method of treatment, as specified by Case Ia, that is, treatment with six radon tubes of 12 millicuries each, inserted for eight days, irrespective of geometrical considerations, indicates that the ureter receives in this case a dose about 25% greater than the minimum dose in the volume treated.

Case Ib is constructed by modifying Case Ia in stages as follows:

1. An additional active length B is added to the active length along the axis of the uterine cavity in order to obtain a decrease in dosage rate from y = 4·0 centimetres to x = 3·5 centimetres.

2. The strength of B relative to that of the remaining components is adjusted to make x = 3·5 centimetres receive a dose only about 75% of that at y = 4·0 centimetres.

3. As a preliminary measure, the strengths of all the components are adjusted in proportion to make the dosage rate at y = 4·0 centimetres the same as in Case Ia at any time during the treatment; subsequently the strengths of all the components are increased by a factor of 1·38 in order to increase the overall dosage. This increase, however, does not make the dose received by the ureters as great as that previously received by them in the old method. The values of the linear strengths of the component active lengths are given in Table III.[1]

Cases II and III then follow from Ib by adjustment of the strength of the square to counterbalance the effect at y = 4·0 centimetres of the variation in its size. Similarly, Case IV is the same as Ib with

[1] In addition, the period of irradiation has been reduced to six days twenty-two hours from the previously used value of eight days, in order that one patient may be treated each week with one set of containers. The remaining period of two hours is sufficient for cleaning the pessary and tubes and reloading them with radon. The values given in Table III apply to the strengths of containers which have been finally adopted.

TABLE IV.

Values of R/N for Points Near the Arrangement of Active Lengths Specified in Table III.

Point Considered. (See Figure I.)	Ia.	Ib.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.
x=1·0 cm. ..	31·5	15·7	14·6	13·8	15·6	14·6	13·7	15·9	14·8	14·0	16·8	15·6	14·8	11·4	11·1	10·8
x=2·0 cms... ..	15·1	9·5	9·2	8·8	9·5	9·1	8·8	9·7	9·4	9·1	10·6	10·3	10·0	10·2	10·1	10·0
x=3·0 cms... ..	7·6	5·8	5·8	5·8	5·8	5·8	5·8	5·9	5·9	5·9	6·4	6·5	6·4	6·6	6·7	6·7
x=3·5 cms... ..	6·0	4·7	4·8	4·8	4·7	4·8	4·8	4·8	4·8	4·8	5·2	5·2	5·2	5·3	5·3	5·3
x=4·0 cms... ..	4·7	3·8	3·8	3·9	3·8	3·8	3·9	3·8	3·9	3·9	4·1	4·1	4·1	4·0	4·0	4·0
y=1·0 cm.	18·4	9·6	9·3	8·7	9·5	9·2	8·6	9·7	9·4	8·8	10·2	9·9	9·2	7·2	7·1	6·9
y=2·0 cms... ..	9·5	7·3	7·2	7·1	7·3	7·2	7·1	7·5	7·4	7·3	8·3	8·2	8·1	9·0	9·0	9·0
y=3·0 cms... ..	6·1	6·9	6·8	6·8	6·9	6·8	6·8	7·1	7·1	7·0	7·8	7·8	7·7	9·2	9·2	9·2
y=4·0 cms... ..	4·7	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5	6·5
Ratio of dosage rate at x=3·5 centimetres to that at y=4·0 centimetres	1·28	0·73	0·73	0·73	0·73	0·73	0·73	0·74	0·75	0·75	0·80	0·80	0·80	0·81	0·81	0·81

the strength of the active length A changed to balance its decrease in length.

When Case VII is reached the active length A must be omitted and the strength of B increased accordingly, because the length of the cavity is now 2·0 centimetres shorter than in Case I*b*. The above procedure is continued to Cases XIII, XIV and XV. The last three cases require some additional manipulation of the relative strengths in order to obtain uniformity.

Actual Values of the Dosage Rates.

The values of $\frac{R}{N}$ given in Table IV may be converted to values of R when N is known. The magnitude of N depends, of course, on the screenage used, and its value has been determined by a number of observers.[12][13][14][15][16][17][18][19] It has been taken here as N = 8·3 Röntgen units per milligramme-hour at 1·0 centimetre distance from a point source with a screenage of 0·5 millimetre of platinum. This gives[20] the following figures for different screenages:

Screenage:	0·5	0·8	1·0	1·5	1·8	2·0 millimetres of platinum
N:	8·3	7·9	7·54	6·9	6·43	6·14 Röntgens per mgm.-hr.

Taking the screenage actually used, that is, 1·8 millimetres of platinum, values of the dosage rate, R, have been found from the figures in Table IV and tabulated in Table V for the series of x and y points.

At this stage it is advisable to note the following points:

1. The values given represent the dosage rate, R, at any time during the treatment if radium is used; but if radon is used, they represent the values of R at the time of insertion, and the values at any other time are obtained by multiplying by the decay factor

$$-7{\cdot}49 \times 10^{-3}\ T,$$

where T is the time in hours after insertion.

2. The total dose delivered to any point is given by integration of the instantaneous value of R over the appropriate time interval.

3. If the screenage differs from 1·8 millimetres of platinum, all the values of R are altered in proportion to the values of N as given above.

4. The linear strengths of the component active lengths (Table III) may be changed in any ratio provided that their relative magnitudes are kept constant and the values of R are changed in the same ratio.

Values of the total dose delivered to the various points considered have been found by integration for the method of treatment actually used, in which the time of insertion is six days twenty-two hours. These values are given in Table VI.

Modification in Strength of the Pessary to Allow for Displacement from the Assumed Position.

In some cases the pessary is thrust down by the loss of the fornices by scarring or growth, and a second series of cases has been considered, Cases I*b*′ to XV′, corresponding to Cases I*b* to XV in the normal series. In the second series the pessary is assumed to be displaced downwards through a distance of 1·0 centimetre.

Values of K have been recalculated for the points y = 1·0, 2·0, 3·0 and 4·0 centimetres, and x = 3·5 centimetres for each of the three sizes of pessary, and it has been found that increasing the strength of the 3·5 centimetre pessary by a factor of 1·63 just counterbalances the effect at the ureter (x = 3·5 centimetres) of its displacement. This factor is 1·59 and 1·57 for the 4·0 and 4·5 centimetre pessaries respectively.

TABLE V.

Initial Dosage Rates (R) at Various Points in Röntgens per Hour (Screenage 1·8 Millimetres of Platinum) for Active Lengths of Strength given in Table III.

Case.	Ia.	Ib.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.
x=1·0 cm.	202	101	94	89	100	94	88	102	95	90	108	100	95	73	71	69
x=2·0 cms.	97	61	59	57	61	59	57	62	60	58	68	66	64	65	65	64
x=3·0 cms.	49	37	37	37	37	37	37	38	38	38	41	42	41	42	43	43
x=3·5 cms.	39	30	31	31	30	31	31	31	31	31	33	33	33	34	34	34
x=4·0 cms.	30	24	24	25	24	24	25	24	25	25	26	26	26	26	26	26
y=1·0 cm.	118	62	60	56	61	59	55	62	60	57	65	64	59	46	46	44
y=2·0 cms.	61	47	46	46	47	46	46	48	48	47	53	53	52	58	58	58
y=3·0 cms.	39	44	44	44	44	44	44	46	46	45	50	50	50	59	59	59
y=4·0 cms.	30	42	42	42	42	42	42	42	42	42	42	42	42	42	42	42

TABLE VI.

Doses in Röntgens Delivered to Various Points for Given Methods of Treatment.

Case.	Ia.	Ib.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.
x=1·0 cm.	19,200	9,600	9,000	8,500	9,500	9,000	8,400	9,700	9,000	8,600	10,300	9,500	9,000	6,900	6,800	6,600
x=2·0 cms.	9,200	5,800	5,600	5,400	5,800	5,600	5,400	5,900	5,700	5,500	6,500	6,300	6,100	6,200	6,200	6,100
x=3·0 cms.	4,700	3,500	3,500	3,500	3,500	3,500	3,500	3,600	3,600	3,600	3,900	4,000	3,900	4,000	4,100	4,100
x=3·5 cms.	3,700	2,900	3,000	3,000	2,900	3,000	3,000	3,000	3,000	3,000	3,100	3,100	3,100	3,200	3,200	3,200
x=4·0 cms.	2,900	2,300	2,300	2,400	2,300	2,300	2,400	2,300	2,400	2,400	2,500	2,500	2,500	2,500	2,500	2,500
y=1·0 cm.	11,200	5,900	5,700	5,300	5,800	5,600	5,200	5,900	5,700	5,400	6,200	6,100	5,600	4,400	4,400	4,200
y=2·0 cms.	5,800	4,500	4,400	4,400	4,500	4,400	4,400	4,600	4,600	4,500	5,000	5,000	5,000	5,500	5,500	5,500
y=3·0 cms.	3,700	4,200	4,200	4,200	4,200	4,200	4,200	4,400	4,400	4,300	4,800	4,800	4,800	5,600	5,600	5,600
y=4·0 cms.	2,900	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000	4,000

When the above adjustments are made, the doses at the points $y = 1{\cdot}0$ and $2{\cdot}0$ centimetres are slightly reduced and those at $y = 3{\cdot}0$ and $4{\cdot}0$ centimetres are slightly increased. Using the modified strengths of pessary ($m = 4{\cdot}07$ millicuries per centimetre for each of the four active lengths D, E, F_1 and F_2 for Cases I*b*′ to XII′ inclusive, and 1·11 millicuries per centimetre for Cases XIII′, XIV′ and XV′), modified values of $\frac{R}{N}$ have been calculated for all the cases used in practice. These values are given in Table VII. Corresponding values of the total dose for the method of treatment actually used are given in Table VIII.

The Construction of Radon Tubes for Practical Application.

To apply to practical cases the dosage system developed in the preceding sections, sets of suitable radon containers and pessaries are necessary. Actually six gold tubes, of the dimensions set out below, are sufficient to cover all cases, since the long inner tube may be allowed to project through the plane of the square pessary for a distance of up to 2·0 centimetres; this projecting portion is not filled with radon. The tubes required for a set are as follows:

One tube:

External length	85·0 millimetres
Internal length	78·0 millimetres
External diameter	4·75 millimetres
Internal diameter	1·75 millimetres

One tube:

External length	55·0 millimetres
Internal length	48·0 millimetres
External diameter	4·75 millimetres
Internal diameter	1·75 millimetres

Four tubes:

External length	28·0 millimetres each
Internal length	21·0 millimetres each
External diameter	4·75 millimetres each
Internal diameter	1·75 millimetres each

One end of each tube is rounded over and has a small hole through which passes a length of number 30 gauge linen thread, which is prevented from being pulled out by a knot at its inside end; the other end of the tube is also rounded and is fitted with a small brass screw so that the tube may be emptied and refilled with radon each week.

Four short tubes are used for each type of case, in conjunction with one of the longer tubes: the 7·8 centimetre tube for Cases I*b* to IX and I*b*′ to IX′ inclusive, and the 4·8 centimetre tube for Cases X to XV and X′ to XV′ inclusive.

The tubes are filled with lengths of radon-filled gold capillary tubing, of screenage 0·3 millimetre of platinum equivalent; in the long tube the three individual active lengths are spaced with short pieces of number 17 gauge copper wire in order to prevent the various sections from overlapping. The radon-filled capillary tubing available may not, of course, be of the correct linear strength as set out in Table III, and additional use is therefore made of copper wire packing to obtain the desired average linear strength for any given active section of the tube in a manner similar to that already fully described for radon needles.[21] In Table IX are given details of the filling of the various component tubes for each type of case.

Construction of Pessaries.

In addition to the radon tubes a set of three pessaries is necessary. Each pessary is made up of four cylindrical tubes of vulcanite set on a solid vulcanite base in the shape of a hollow square; the lateral and inferior edges of the square are cut away. The distance between the axes of the tubes on any two opposite sides of the square is 3·5, 4·0 and 4·5 centimetres for the three sizes employed. One of the pessaries is illustrated in Figure IV. A lead mantle of a thickness of 8·0 millimetres covers the dorsal half of the rear cylinder and helps to

TABLE VII.

Value of R/N Modified by the Use of Displaced Pessaries.

Point Considered. (See Figure I.)	I*b*′.	II′.	III′.	IV′.	V′.	VI′.	VII′.	VIII′.	IX′.	X′.	XI′.	XII′.	XIII′.	XIV′.	XV′.
x=3·5 cms.	4·7	4·8	4·8	4·7	4·8	4·8	4·8	4·8	4·8	5·1	5·2	5·2	5·3	5·3	5·3
y=1·0 cm.	7·8	7·6	7·3	7·8	7·5	7·3	7·9	7·7	7·4	8·6	8·2	7·9	6·7	6·6	6·6
y=2·0 cms.	7·2	7·1	6·9	7·1	7·0	6·9	7·4	7·2	7·1	8·1	8·0	7·9	9·0	8·9	8·9
y=3·0 cms.	7·0	6·9	6·9	7·0	6·9	6·9	7·2	7·1	7·1	7·9	7·8	7·8	9·2	9·2	9·2
y=4·0 cms.	6·8	6·6	6·5	6·8	6·6	6·5	6·8	6·6	6·5	6·8	6·6	6·5	6·5	6·5	6·5
Ratio of dosage rate at x=3·5 centimetres to that at y=4·0 centimetres	0·70	0·72	0·73	0·70	0·72	0·73	0·71	0·73	0·74	0·76	0·79	0·79	0·80	0·81	0·81

TABLE VIII.

Doses in Röntgens Delivered at Various Points with a Given Method of Treatment.
(Pessary Displaced.)

Case.	I*b*′.	II′.	III′.	IV′.	V′.	VI′.	VII′.	VIII′.	IX′.	X′.	XI′.	XII′.	XIII′.	XIV′.	XV′.
x=3·5 cms.	2,900	2,900	2,900	2,900	2,900	2,900	2,900	2,900	2,900	3,100	3,200	3,200	3,300	3,300	3,300
y=1·0 cm.	4,800	4,700	4,500	4,800	4,600	4,500	4,800	4,700	4,500	5,300	5,000	4,800	4,100	4,100	4,100
y=2·0 cms.	4,400	4,400	4,200	4,400	4,300	4,200	4,500	4,400	4,400	5,000	4,900	4,800	5,500	5,500	5,500
y=3·0 cms.	4,300	4,200	4,200	4,300	4,200	4,200	4,400	4,400	4,400	4,800	4,800	4,800	5,600	5,600	5,600
y=4·0 cms.	4,200	4,100	4,000	4,200	4,100	4,000	4,200	4,100	4,000	4,200	4,100	4,000	4,000	4,000	4,000

TABLE IX.
Data for the Filling of Radon Tubes.

Case.	Filling of the Long Axial Tube.								Four Tubes of 2·0 Centimetres.		Total Radon Used in Millicuries.
	Internal Length in Centimetres.	Length Spacing at Thread End in Centimetres.	Thread End.		Centre Part.		Screw End.		Each in Millicuries per Centimetre.	Each in Millicuries.	
			Centimetres.	Millicuries.	Centimetres.	Millicuries.	Centimetres.	Millicuries.			
Ib	7·8	0	2·0	5·02	3·7	18·5	2·0	5·02	2·51	5·02	48·6
II	7·8	0	2·0	5·02	3·7	18·5	2·0	5·02	2·56	5·13	49·1
III	7·8	0	2·0	5·02	3·7	18·5	2·0	5·02	2·61	5·22	49·4
IV	7·8	1	2·0	5·02	3·7	18·5	1·0	3·96	2·51	5·02	47·6
V	7·8	1	2·0	5·02	3·7	18·5	1·0	3·96	2·56	5·13	48·0
VI	7·8	1	2·0	5·02	3·7	18·5	1·0	3·96	2·61	5·22	48·4
VII	7·8	2	2·0	5·02	3·7	20·8	—	—	2·51	5·02	45·9
VIII	7·8	2	2·0	5·02	3·7	20·8	—	—	2·56	5·13	46·3
IX	7·8	2	2·0	5·02	3·7	20·8	—	—	2·61	5·22	46·7
X	4·8	0	2·0	5·02	2·7	20·8	—	—	2·51	5·02	45·9
XI	4·8	0	2·0	5·02	2·7	20·8	—	—	2·56	5·13	46·3
XII	4·8	0	2·0	5·02	2·7	20·8	—	—	2·61	5·22	46·7
XIII	4·8	1	2·0	1·36	1·7	29·4	—	—	0·681	1·36	36·2
XIV	4·8	1	2·0	1·36	1·7	29·4	—	—	0·697	1·39	36·3
XV	4·8	1	2·0	1·36	1·7	29·4	—	—	0·710	1·42	36·4
Ib′	7·8	0	2·0	5·02	3·7	18·5	2·0	5·02	4·07	8·14	61·1
II′	7·8	0	2·0	5·02	3·7	18·5	2·0	5·02	4·07	8·14	61·1
III′	7·8	0	2·0	5·02	3·7	18·5	2·0	5·02	4·07	8·14	61·1
IV′	7·8	1	2·0	5·02	3·7	18·5	1·0	3·96	4·07	8·14	60·0
V′	7·8	1	2·0	5·02	3·7	18·5	1·0	3·96	4·07	8·14	60·0
VI′	7·8	1	2·0	5·02	3·7	18·5	1·0	3·96	4·07	8·14	60·0
VII′	7·8	2	2·0	5·02	3·7	20·8	—	—	4·07	8·14	58·4
VIII′	7·8	2	2·0	5·02	3·7	20·8	—	—	4·07	8·14	58·4
IX′	7·8	2	2·0	5·02	3·7	20·8	—	—	4·07	8·14	58·4
X′	4·8	0	2·0	5·02	2·7	20·8	—	—	4·07	8·14	58·4
XI′	4·8	0	2·0	5·02	2·7	20·8	—	—	4·07	8·14	58·4
XII′	4·8	0	2·0	5·02	2·7	20·8	—	—	4·07	8·14	58·4
XIII′	4·8	1	2·0	1·36	1·7	29·4	—	—	1·11	2·22	39·6
XIV′	4·8	1	2·0	1·36	1·7	29·4	—	—	1·11	2·22	39·6
XV′	4·8	1	2·0	1·36	1·7	29·4	—	—	1·11	2·22	39·6

protect the rectum, and by its weight tends always to remain *in situ* when the patient is in the dorsal decubitus position. At the time of removal the pessary will always be found in the correct rotational position.

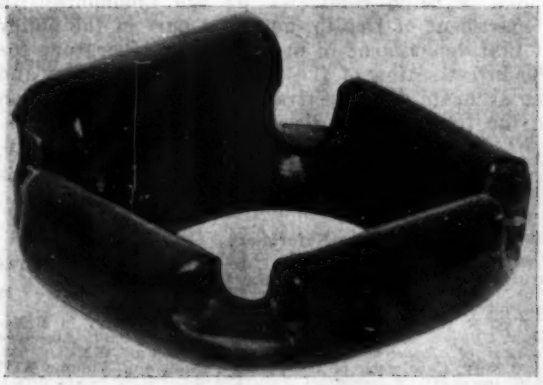

FIGURE IV.
Vulcanite pessary.

Clinical Application.

In most methods of irradiation of cervical cancer with γ rays an intrauterine tube or series of tubes in tandem is used with some form of vaginal applicator.[3][4][5][6][7] The intrauterine tube is usually inserted after dilatation of the cervix, and the vaginal applicator (a colpeurynter and various types of pessary could be included under this term) is usually held in position with packing. Packing is also used to keep the rectum and bladder away from the sources of irradiation. In the present method the central intrauterine container is used covered with rubber; as has already been indicated, its length is varied as well as the distribution and strength of radon along its length. The square

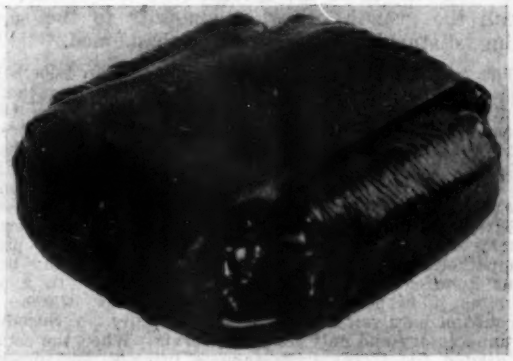

FIGURE V.
Pessary loaded for use.

pessary used, as described above, contains the four short gold tubes in the four vulcanite cylinders around its sides, the gold tubes being retained in position by rubber bands. Broad rubber bands (see Figure V) are crossed over the pessary in a cruciform manner to form a platform on which the end of the intrauterine tube rests when in position. The pessary lies in the fornices and its sloping sides rest on the inclined planes of the *levatores ani* muscles. Action of these muscles therefore tends to drive the pessary into the fornices and against

the cervix. No packing of any kind is used; packing brings the radon in the left fornix in closer relation to the rectum than when none is used. Since the adoption of the pessary with the lead mantle and no packing, even mild proctitis has not been seen.

The length of the uterus is measured with an ordinary graduated uterine sound. The size of pessary to be used is the largest possible pessary that can be got to lie in apposition to the growth. In ordering radon for treatment, the size of pessary is specified and the length of the uterine tube; from this data the appropriate case, as set out in Table IX, may be selected and the containers prepared accordingly.

In treatment preliminary rest in bed is essential. No effort is made to clean the growth, except by the use of saline douches. Chemical disinfectants should always be avoided. Rest and a saline douche clean the growth up as well as any other measure, and it is better to treat a grossly infected lesion than to waste too much time trying to clean it up.

Patients with marked anæmia (below 40% hæmoglobin) should not be treated until, by repeated small blood transfusions combined with the usual medical treatment, their hæmoglobin index has been raised. Time and trouble spent in this are well repaid.

Patients with a high blood urea content should never be treated with radon without previous medical treatment. They are very liable to become uræmic, even when the greatest care is exercised. They require constant attention after treatment, and even at their best their prognosis is poor.

Serious infection has become rare since efforts to clean the growth or, what amounts to the same thing, disturbing it, have been abandoned.

The vagina is gently swabbed out with a 1 in 1,000 solution of acriflavine. Many patients require no anæsthetic. For some years now a long hypodermic needle has been inserted into each lateral vaginal fornix and about ten cubic centimetres of a 2% solution of "Novocain" have been injected. The needle is inserted until the bulge of the side of the uterus is felt and then withdrawn about 1·25 centimetres (half an inch). The fluid is then injected in about the region of the crossing of the ureter and the uterine artery—the region in which the sympathetic and parasympathetic fibres of the cervix cross. This has been done hundreds of times without any trouble. In a few minutes it is possible to dilate the cervix, which, on account of paralysis of muscle, dilates easily. A volsellum is unnecessary and only tears the cervix. When the cervix is dilated the intrauterine tube is inserted and pressed up to the top of the fundus. The pessary is then inserted and pushed gently into the fornices, care being taken to see that the lead-covered tube lies in the posterior fornix and that the end of the intrauterine tube lies on the top of the crossed broad rubber bands. No packing is used.

The patient is kept in the dorsal decubitus position for the treatment period of six days twenty-two hours. No catheter is used and the bowels are opened at regular intervals. Most patients have some rise in temperature, but no rise in pulse rate, and suffer no discomfort during the period of treatment.

There has not been any serious proctitis since the lead mantle was used. The use of aloes and cascara should be avoided, as it gives rise to some very serious pain. Liquid paraffin is the only aperient which can be used with safety in all cases. A proctoscope will often show some deepening of colour of the anterior rectal wall, even in those patients with no clinical symptoms of proctitis. It is in these cases that aperients cause severe pain. Only morphine will stop the pain of proctitis.

The rare radium cystitis is apt to be mistaken for a recurrence; but the cystoscopic appearances are quite distinct. The florid recurrence should never be mistaken for the pale areas with flat ulceration seen in the so-called radium cystitis. It is apparently an arteriosclerotic condition. The majority of cases clear up in time with saline irrigation. Strong solutions, particularly of silver nitrate, are apt to cause grave sloughing.

Summary.

A method is developed for the treatment of cervical cancer by means of radon tubes, the radioactive strengths of which have been chosen with the object of providing comparable dosage distributions throughout the affected region adaptable to different cases in spite of varying geometrical factors. Consideration has also been given to the avoidance of overdosage of surrounding viscera while an adequate amount of radiation to the affected area is delivered.

A description is given of the applicators and clinical method employed.

Acknowledgements.

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CHOLINE ESTERASE IN MYASTHENIA GRAVIS.

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IN view of recent evidence that the transmission of the nerve impulse to voluntary muscle is possibly mediated by acetylcholine, and, further, that human blood contains a specific esterase that rapidly hydrolyses this substance, it has been suggested that the muscular weakness of *myasthenia gravis* might be due to an increased activity of blood esterase. This would imply that, owing to excessively rapid destruction of acetylcholine, the muscles would not receive an adequate stimulus and would, therefore, fail to contract in a normal manner. Eserine is known to inhibit esterase activity and should, therefore, relieve the symptoms of *myasthenia gravis*. Actually, relief occurs in a most dramatic fashion, especially when the non-toxic derivative, "Prostigmin", is used.

Against such an attractive theory we must remember that: (i) no generalized parasympathetic disturbances, as would be expected to follow an increased esterase activity in the circulating blood, are found in *myasthenia gravis*; (ii) frequently the muscular weakness is limited to a group of muscles.

Determinations of the choline esterase activity in the blood of myasthenic patients have produced conflicting results. Thus Stedman[1] and McGeorge[2] found no increased activity, while Hicks,[3] from a prolonged study on a patient with marked myasthenic symptoms, invariably found an increased esterase activity in the serum.

The paper by McGeorge appeared whilst our own investigations were in progress, and his conclusions entirely agree with ours, namely, that marked variations occur in the blood of normal individuals and also in patients with various medical conditions. Furthermore, in the latter group there appears to be no correlation between esterase activity and the clinical condition of the patient. This statement applies also to patients suffering from *myasthenia gravis*.

In our own experiments we have studied two patients with pronounced symptoms of *myasthenia gravis* for periods of two and eight months respectively.

Methods.

In our earlier experiments we estimated acetylcholine by means of the rabbit's intestine, according to the technique described by Dale[4] and Matthes.[5] Later, at the suggestion of Dr. Feldberg, we abandoned this method for the one in which the frog's rectus muscle is used (Chang and Gaddum[6]).

For the determination of esterase activity, blood containing potassium oxalate as an anti-coagulant was allowed to act on a standard solution of acetylcholine (Hoffmann-La Roche) for exactly 20 seconds at 12° C. Esterase action was then stopped by the addition of a 20% solution of trichloracetic acid, which also coagulated the blood proteins. Trichloracetic acid was removed from the protein-free filtrate by successive shakings with ether in a separatory funnel, after which the ether was removed by aeration. Suitable dilutions were then made from this solution and compared in respect of acetylcholine concentration with a standard solution. Estimations were made on the frog's rectus muscle, which was suspended in a glass bath containing approximately three cubic centimetres of Ringer solution. In order to potentiate the effect of acetylcholine, eserine to give a final concentration of 1 in 100,000 was added to the Ringer solution. All estimations were carried out by comparing the actions of the test and standard solutions on the muscle strip after a period of exactly two minutes. The actual quantities of substances used in preparing a blood filtrate for estimating choline esterase activity are shown in Table I.

TABLE I.

Substance.	Cubic Centimetres.
Ringer solution (Locke's solution diluted 1 in 1·3) ..	2·5
Blood	0·5
Acetylcholine 1/10,000	1·0
Trichloracetic acid 20%	1·0
Total	5·0

The final concentration of acetylcholine in the above filtrate should be 1 in 50,000; but, owing to esterase action, it will be less. From the actual value found can be calculated the percentage destruction under the stated experimental conditions. With this method it is necessary to be sure that none of the observed effect of the test solution in causing a contraction of the rectus

muscle is due to potassium. That this might quite possibly occur will be realized when it is remembered that potassium oxalate was used as an anticoagulant, and also that the estimations were made on whole blood, the red blood corpuscles of which contain appreciable quantities of potassium salts. In order to assure ourselves that this error was not present, the following controls were carried out:

1. Blank experiments on the lines shown in Table I were carried out. In these, water containing the same quantity of potassium oxalate was substituted for the blood, whilst the other constituents remained constant. The final solution showed an acetylcholine content that closely corresponded to the theoretical value, that is, 1 in 50,000. From this we concluded that no effect was being produced on the muscle contraction by the potassium present as potassium oxalate.

2. After an estimation of acetylcholine had been made on a test solution, the rectus muscle was suspended for 30 minutes in Ringer solution containing atropine. It was then found that the test solution had no significant effect on the muscle, thus showing that the observed contraction was really an acetylcholine effect. In this experiment any error due to potassium present either in the red blood corpuscles or as added oxalate was eliminated.

We have calculated that the total potassium content of our test solutions was at the maximum about 1·7 milligrammes per ten cubic centimetres, and experimentally we found that from seven to ten milligrammes of potassium per ten cubic centimetres of fluid were required to cause a definite effect on the rectus muscle.

Results.

As previously stated, our observations on the two myasthenic patients extended over a considerable period. Usually tests were made when the symptoms of muscular weakness were most pronounced. In some instances daily estimations were made for two weeks. In addition, blood samples for estimation of choline esterase activity have been taken from a number of normal individuals and from patients in the medical and surgical wards of the hospital. In general, we can confirm the view expressed by others that the choline esterase activity of the blood of the same individual remains fairly constant. Great variations were found in different individuals, both normal and otherwise. The differences found appeared to bear no relation to the clinical condition of the patient, and, furthermore, the values found in the myasthenic patients fell well within the normal range (see Table II).

TABLE II.

Number.	Patient's Condition.	Percentage Destruction of Acetylcholine.
1	*Myasthenia gravis.* Pronounced symptoms	50
2	*Myasthenia gravis.* Pronounced symptoms	57
3	Normal	60
4	Normal	64
5	Normal	38
6	Normal	61
7	Fractured femur	76
8	Fractured femur	58
9	Fractured humerus	66
10	Rheumatoid arthritis. No temperature	50
11	Normal	72
12	Normal	73
13	Chronic nephritis	63
14	Chronic nephritis	53
15	Chronic nephritis	47
16	Normal	44
17	Normal	52
18	Headaches, six months after head injury	29
19	Cardiac failure	50
20	Cardiac failure	43
21	Cardiac failure	51
22	Cardiac failure	54
23	Normal	60
24	Normal	46
25	Normal	77
26	Normal	60
27	Normal	52
28	Normal	47

In Figures I and II the results of experiments on a patient are shown. This man was admitted to the hospital with severe symptoms of *myasthenia gravis*. Blood was collected at a time when extreme muscular weakness was present. In Figure I it is shown that a dilution of the filtrate prepared from the patient's blood (see Table I) has an acetylcholine concentration that lies between 1 in 11,000,000 and 1 in 10,000,000 of a standard solution. In this case the test solution prepared from the patient's blood was diluted, 0·1 cubic centimetre in 11 cubic centimetres of Ringer solution being used, and the percentage destruction was calculated as approximately 50.

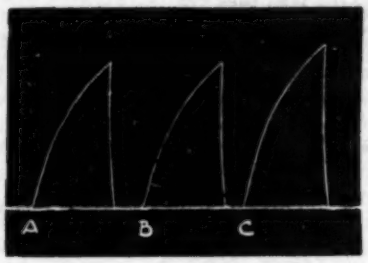

FIGURE I.

Estimation of choline esterase in blood of myasthenic patient. Test on frog's rectus muscle. *A.*: response to standard acetylcholine solution 1 in 11,000,000; *B.*: response to test solution (from patient's blood) diluted 0·1 cubic centimetre in 11·0 cubic centimetres Ringer solution; *C.*: response to acetylcholine 1 in 10,000,000 (destruction 50%).

In Figure II the effect of "Prostigmin" on this patient is shown. Blood was taken one hour after the injection of this drug, and it is seen that the percentage destruction is now reduced to 19. In contrast to this patient, the result of an experiment on a perfectly healthy individual is shown in Figure III, and it will be observed that, under

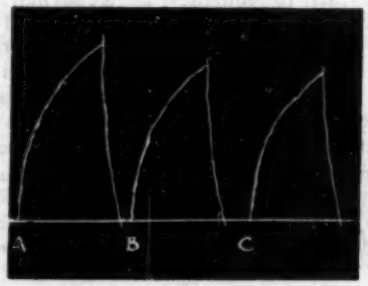

FIGURE II.

Tests on same patient as in Figure I, but one hour after the administration of "Prostigmin". *A.*: response to acetylcholine 1 in 10,000,000; *B.*: response to test solution, diluted 0·1 cubic centimetre in 17·0 cubic centimetres; *C.*: response to acetylcholine 1 in 11,000,000 (destruction 19%).

the same experimental conditions as were present in Figure I, the estimated acetylcholine destruction was 73%. On several occasions the test was repeated on this normal individual and high values were always obtained.

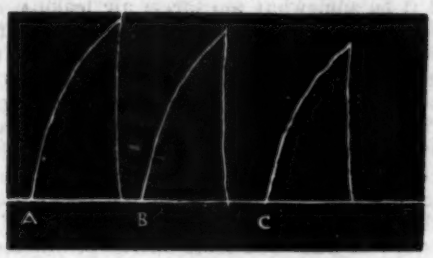

FIGURE III.

Estimation of esterase activity in normal individual. *A.*: response to acetylcholine 1 in 12,000,000; *B.*: response to test solution 0·1 cubic centimetre in 6·0 cubic centimetres Ringer solution; *C.*: response to acetylcholine 1 in 13,000,000 (destruction 76%).

In Table II the values obtained for choline esterase activity in a number of individuals are shown. It is expressed as percentage destruction of acetylcholine added to blood according to the conditions shown in Table I.

These figures indicate the wide variations that we found in estimating choline esterase activity. In Case 18 a very low value was found, but on repeating the test this finding was confirmed.

Comment.

It seems certain that the condition of muscular weakness in the two patients studied cannot be accounted for by an increased choline esterase activity of the blood. It might be added that in both patients a pronounced temporary relief of the symptoms followed the administration of "Prostigmin". However, despite this, it seems that for the time being the only safe assumption is that of McGeorge, namely, that there is either a deficient production of acetylcholine at the neuro-muscular junction, or that the local esterase activity is increased. In this connexion Hicks has also suggested that the increased values that he found in the serum might have been due to an "overflow" of a tissue esterase.

Acknowledgement.

We wish to thank Dr. L. B. Cox for permitting us to carry out the necessary investigations on the patients under his charge.

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THE TREATMENT OF THE RESPIRATORY FAILURE OF POLIOMYELITIS.

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DURING the present outbreak of poliomyelitis in Melbourne there have been many inquiries from country districts concerning types of respirators and management of patients in them.

Since late in July of this year all patients with poliomyelitis in and about Melbourne have been admitted to Fairfield. Before the commencement of the outbreak we had had one Drinker respirator in use and immediately took over two other machines which were being constructed for the Children's Hospital, where patients had previously been treated. Now, four months later, we have twenty-three in use, and we have lately installed six constructed under the supervision of the engineer to the Health Department of Victoria. It is expected that machines of similar type will be made for Victorian country districts.

All those in use here work on the principle of a negative pressure expanding the chest wall, resulting in inspiration, followed by a return to atmospheric pressure, resulting in expiration. The advantages of this method will be understood by anyone who has tried manually to produce artificial respiration on a child with intercostal paralysis. The essential rebound of the ribs is lacking to some extent when intercostal paralysis is present, and one finds difficulty in producing much movement of the chest wall. For this reason there is considerable doubt whether respirators which depend for their action on positive pressure compressing the chest could be of much use in intercostal paralysis. At the time of writing 79 patients have received respirator treatment, and of these, 26 have died. Of those still living, it would be a mistake to suppose that all owe their lives to the aid of the respirator, but undoubtedly the majority do so. In most of the fatal cases death was due to causes other than respiratory: twenty-one patients had marked pharyngeal involvement. Thirteen of the twenty-six deaths occurred within forty-eight hours of the commencement of respirator treatment.

The respirator treatment of respiratory failure has many difficulties, and we shall enumerate and discuss some of the problems that we have encountered so far in this epidemic: (i) the indications for its use, (ii) the technique of placing the patient in the respirator to ensure the minimum of distress, (iii) the nursing and management of the

patient while in the respirator, (iv) the treatment of patients with bulbar involvement, (v) the indications for cessation of respirator treatment.

The Indications for the Use of the Respirator.

The diagnosis of respiratory failure must first be established, and differentiated from such conditions as pneumonia or obstruction due to mucus. Paralysis of the intercostals alone is shown by lessened movement of the thorax and increased movement of the abdomen. Commonly there is a reversal of the normal thoracic movement, and as the diaphragm descends with inspiration the thorax actually retracts instead of expanding. In paralysis of the diaphragm alone there is increased movement of the intercostals with a consequent retraction of the abdomen when this becomes pronounced. Involvement of the intercostals completely and of the diaphragm partially, or *vice versa*, will be shown by increased use of the accessory muscles of respiration. The *alæ nasi* will be working, and the action of the platysma causes movement of the neck and chin.

At present we withhold respirator treatment until it is certain that paralysis is definitely extending and the patient is showing some signs of distress, such as pallor, restlessness and sweating; cyanosis is a sign of urgency and should not be allowed to develop. When placed in the respirator a child who has been distressed will respond immediately; the breathing rate will synchronize with the machine, and the child will sleep peacefully—the relief is dramatic in most cases. It seemed at first that respirator treatment should be instituted at the first signs of respiratory weakness. It was hoped that in this way complete rest would be given to weakened muscles, and that there would be lessened stimulation to diseased nerve cells. Further consideration and observation cast doubt on the degree of muscle rest thus afforded. Certainly the breathing becomes largely a passive movement as compared with the normal active movements, but in the respirator the degree of actual excursion is increased. Observation of children with intercostal paralysis shows what surprisingly little chest movement is needed to maintain comfort and a good colour. Further, it has been shown by Brahdy and Lenarsky[1] that respirator treatment may in itself be responsible for some pulmonary complications. We have seen two fatal cases in which there was extensive surgical emphysema of one lung, the mediastinum and the abdominal cavity. Consequently we delay treatment until it is seen to be necessary.

The Technique of Placing the Patient in the Respirator.

The most difficult part is the fitting of the rubber neckpiece in position, and this is most easily done with the patient lying in the open respirator. The back of the neck is first protected with strips of "Elastoplast", a procedure we have found of great value for parts which are exposed to any pressure. The neckpiece is made of soft sponge rubber, proofed on one side with a thin sheet of impervious rubber. Care should be taken to ensure that the hole for the neck is of such a size that it fits easily and without causing any obstruction to breathing; should it be somewhat too large, air leakage round the neck can be stopped by cotton wool. To fit the neckpiece in position, the operator passes both hands through the hole for the neck and places one hand over the patient's occiput and the other over the face. Two assistants then stretch the neckpiece over the hands so that it need not come into rough contact with any part of the patient's face. The patient is lifted so that the head, with the neckpiece now in position, is carried through the head opening in the respirator and then rests on the pillow outside. The collar is now clamped in place; its movement with respiration may be minimized by placing metal plates or bars across the head opening. Sponge rubber pads are placed between the shoulders and the front of the respirator, and the skin of the

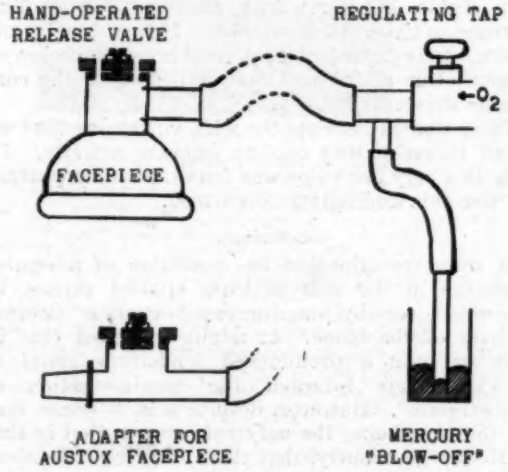

FIGURE I.

shoulder points may also be protected with "Elastoplast". When the respirator is started, it may be necessary for a time to encourage the child to synchronize with the machine. Reassurance and the indication of the appropriate moments for inspiration and expiration are all that is necessary. Occasionally a child will attempt to breathe faster than the respirator rate. We have found that in these cases it is a mistake to increase the rate of the machine. If the patient does not soon synchronize, then either he does not need the respirator, which is unlikely, or the prognosis is very unfavourable. Similarly, when a child who has been breathing in step with the respirator for some time loses his synchronism, a fatal termination may soon be expected. It is difficult to explain this irregularity of the breathing in any other way than by irritation of the respiratory centre. We have found that the most comfortable breathing rate is about 20 to 24 per minute, with a negative pressure of about 20 centimetres of water.

The Nursing and Management of the Patient in the Respirator.

The following points will be discussed: (*a*) the position of the patient, (*b*) feeding, (*c*) changing the patient, (*d*) splinting.

The Position of the Patient.—All our respirators can be tilted so that the head can be raised or lowered. This is essential in the treatment of pharyngeal paralysis. A side-to-side tilting was considered desirable, but as this required a complicated mechanism, we have not installed it on any of our machines. If hypostatic pneumonia is feared the position of the patient may be changed to some extent by placing pillows on one or other side of the body.

done by the nursing staff, and only occasionally is the presence of a doctor required. The apparatus is simple and consists of a gas machine facepiece in which is incorporated a manually controlled valve to release pressure, and a mercury "blow-off" of 10 to 15 millimetres, which acts also as a pressure gauge. An adequate stream of oxygen, of a volume soon found by experience, is delivered under pressure to the patient, the lungs being thus inflated, and then, by means of the release valve, deflated. This is readily followed by watching the pressure gauge, and the hand valve is operated rhythmically at about twenty times a minute. The "blow-off" safeguards the lungs against undue pressure, but, on the other hand, leakage at the facepiece must

FIGURE II. A portion of respirator ward.

Feeding.—No difficulty has been experienced in feeding respirator patients, with the exception of those with pharyngeal involvement. There is no need to stop the respirator during feeding. The patient automatically closes the glottis and food is taken readily. With fluids it is desirable to use a straw or glass tube so that the patient can adjust his own rate of swallowing. Most of the children who have been in the respirator for weeks have excellent appetites.

Changing the Patient.—Many of those with paralysis of the intercostals and diaphragm cannot do without the respirator for more than a few seconds, and even when one of the side ports is opened may become cyanosed. The changing and washing of such patients at first caused endless worry. A method worked out by one of us (J.W.) has completely altered this. The changing is now

be reduced to a minimum in the interests of efficiency. The usual "Austox" facepiece may be used with the addition of a release valve of the type illustrated. Considerable respiratory ventilation may be maintained in this way for periods ample to fulfil all nursing requirements, and the patient's colour remains all that could be desired.

Splinting.—No elaborate splinting is attempted while the patient needs continuous respirator treatment. Plaster cases for the hands, forearms and lower limbs are all that can be managed. As the deltoids are of necessity in a bad position in the respirator, it is desirable that some movements be commenced as soon as possible. When the patient is able to do without the respirator for six to eight hours he is fitted with a double Thomas splint with armpieces, which is worn during that time.

The Treatment of Respirator Patients with Bulbar Involvement.

Patients with bulbar involvement may be divided, purely on the basis of mode of termination, into three groups: (*a*) those with involvement of the pharyngeal muscles, (*b*) those with involvement of the "vital" (cardiac and respiratory) centres only, (*c*) those in whom both pharyngeal muscles and vital centres are involved.

The outcome in the last two groups has been rapidly fatal. The termination is sudden, often dramatic, and treatment with oxygen and "Coramine" or by increasing the respirator rate has been of no avail. As previously stated, persistent failure of the patient to synchronize his breathing with the respirator is a bad prognostic sign, and it may be mentioned that "coffee-ground" vomitus, which sometimes appears, is of equal significance.

Therefore the treatment of patients in the first group will be considered, that is to say, of patients with respiratory paralysis and involvement of the pharyngeal muscles. It may be stated at the outset that so far all of our cases of this type have been severe and the patients have died within three weeks; but as the treatment also applies to patients suffering from pharyngeal paralysis without respiratory failure, it will be described.

In the acute stage of the condition, mucus in the pharynx, often becoming frankly purulent, is very troublesome, and no doubt contributes greatly to the production of pulmonary sequelæ. It is dealt with as far as possible by the use of the steam or water sucker, which seems to be a most valuable measure and should be used frequently. We have not found an indwelling sucker to be practicable with the tenacious mucus which is encountered, nor to be well tolerated by the child, who is usually very restless at this stage. In addition, the patient is tilted, head downward, at an angle of 15° to 20°; repeated doses of atropine given hypodermically may be found beneficial.

In all our combined "respiratory and pharyngeal" cases the inability to swallow has been marked, and vomiting and regurgitation may occur. No oral fluids are given in the first forty-eight hours, and this applies also to nasal feeding, which may be, during this early period, a distressing and dangerous procedure. Use of the intravenous route is difficult in a respirator, and no advantage is gained as a rule. Fluids are administered rectally, either by repeated injection or by a continuous drip apparatus. As mentioned above, restlessness or even delirium is common and the exhibition of sedatives becomes necessary. Chloral and bromide may be given rectally, but at times morphine is required and cannot be considered harmful in respirator cases. Aspiration pneumonia and atelectasis often occur, and then the administration of oxygen in addition to the above measures may be of use.

The Indications for the Cessation of Respirator Treatment.

There seems to be no doubt that the patient should be encouraged to breathe naturally as soon as possible. Children especially are too ready to allow the work to be done for them; added to this is a further considerable factor—they become fearful of what will happen when the respirator stops.

When it is found that the patient can be changed without oxygen he is encouraged to go a little longer. Rewards may be offered, or the competitive spirit induced between patients at about the same stage will often produce astonishing results. Another method in obstinate cases is to reduce gradually the negative pressure of the machine until the patient is doing most of the work for increasing periods. At this stage, however, a word of warning may be necessary. Signs of actual fatigue should be watched for carefully; pallor and sweating are indications of danger. Should collapse occur, it may be the commencement of a steady decline in the condition of the patient; so that at this time one should make a study of each individual. Those who are fearful should be encouraged by rewards and subterfuge, while those who are obviously trying hard should be watched carefully and restrained if necessary. It may be mentioned in passing that we have two children who have been in respirators for eleven weeks and who still cannot be changed without oxygen. Their future appears to be gloomy, yet in the respirator they seem to be bright, normal children. On the other hand, two others who have been in for the same length of time have succeeded during the last week in doing without the respirator for half an hour daily.

Our present method is to keep the child continuously in the respirator, except for changing, during the first week. If then oxygen is not necessary, the period out of the respirator is increased up to half an hour and doubled on each succeeding day if this can be accomplished without distress. With the idea of maintaining an efficient aeration of the lungs, we have persisted with a period of four hours' respirator treatment daily for long periods, up to three months in some patients. Breathing exercises are given frequently, the children being encouraged to blow up balloons, to blow trumpets, and so on.

It seems, from Brahdy and Lenarsky's follow-up of respirator cases, that some patients will develop massive atelectasis within a year or two. Any upper respiratory or pulmonary infection will be a serious danger; in very few severe cases are the patients able to give a proper expulsive cough, and pneumonia will probably be of common occurrence. Most of these patients have a persistent slight rise in temperature, perhaps indicating some degree of pulmonitis. However, X ray examinations of sixteen patients have failed to show any evidence of pathological change in the lung. We intend to follow these up with further radiological investigation.

Though the ultimate result may not be satisfactory in some cases, it must be concluded that in those cases of respiratory failure in which there is no bulbar involvement, respirator treatment is of the utmost value.

Acknowledgements.

We wish to record a deep debt of gratitude to Professor Burstall, of the Engineering School of the University of Melbourne, and to the firms which cooperated with him in producing so rapidly the earlier respirators. Our thanks are due to Dr. F. V. Scholes for his valuable help in the preparation of this paper.

Reference.

(1) M. B. Brahdy and M. Lenarsky: "Respiratory Failure in Acute Epidemic Poliomyelitis: Late Results and Complications", *Journal of Pediatrics*, Volume VIII, April, 1936, page 420.

Reviews.

PHARMACOLOGY.

A. J. CLARK'S "Applied Pharmacology" is a book of such outstanding merit that a new edition is welcome. It is more than a text-book for medical students. It is a most valuable work of reference for the library of any medical practitioner. The sixth edition has now been published.[1] As the preface informs us, the most important change in this edition is the collecting together of all the substances which the body obtains normally in its food or manufactures itself. The rapid increase in therapeutic importance of such substances has necessitated that arrangement. It is pointed out that our knowledge of the chemistry of enzymes, hormones and vitamins is advancing with amazing rapidity. Investigation clearly shows the extraordinary diversity of substances grouped together in these classes. However, from the physiological standpoint they display a common quality in that they are all parts of the system of chemical control which regulates the functions of the body. Portions of the work are devoted to the fate of drugs in the body and detoxication of drugs. It is shewn that the defensive mechanisms of the body are not confined to the liver and kidneys, but are numerous and varied. The action of acetylcholine is localized by the presence in the blood of an enterase which rapidly splits the compound. Other matters considered are the relation of chemical constitution to drug action, the hormonal theory of autonomic control and neuro-humoral transmission. Concerning these matters knowledge is ever increasing. Reference is made to the investigations of C. H. Kellaway on cobra venom, published this year. Kellaway believes that this venom acts by liberating histamine in the tissues. Among other new matters dealt with is benzedrine—one of the sympathomimetic drugs. It is pertinently stated that its therapeutic use as a stimulant of the central nervous system is still in the experimental stage and that it may evoke undesirable cardiac effects. In discussing the treatment of *myasthenia gravis* by physostigmine and prostigmine the author states that the mode of action of prostigmine in this disorder is unknown; it is not interfered with by atropine. "Prontosil" and p-sulphonamide have due recognition and critical discussion. Since this work was in the press untoward effects from the administration of these drugs have become more common, and a recent disaster in America awaits complete explanation. "Germanin" (Bayer 205) for trypanosome infections is explained. Mandelic acid therapy is stated to be much superior to any previously known form of urinary disinfection. In discussing endocrine therapy Clark insists that a large proportion of modern organotherapeutics is devoid of any scientific basis and simply represents a reversion to mediæval superstition. Again, he states that whatever be the source of a new preparation, someone is always ready to attribute to it beneficial therapeutic effects. In regard to sex hormones, Clark states that gratifying results are frequently reported from the use of some preparations, but that these effects must be regarded as interesting examples of psychotherapy and have no connexion with the pharmacological action of sex hormones. It is interesting to find that Clark considers that parathyreoid extract produces its action even when given by mouth. A careful study of the whole book is of the highest value.

[1] "Applied Pharmacology", by A. J. Clark, M.C., M.D., F.R.C.P., F.R.S.; Sixth Edition; 1937. London: J. and A. Churchill Limited. Demy 8vo, pp. 688, with 83 illustrations. Price: 18s. net.

PSYCHOANALYSIS.

So many misconceptions exist concerning the method of psychoanalysis that Dorothy Blitzsten's small book, "Psychoanalysis Explained", should be read by any who wish to be better informed.[1] There is, however, one misstatement in it which is too important to be ignored. On page 30, after defining psychoanalysis as a dissection of the pattern of the patient's life, the authoress states: "there follows a re-education in which the analyst leads" (that is, leads the patient). Actually every successful analysis results in an increased interest in life, but only a "pseudo-analyst" would attempt to reeducate his patients; a trained analyst would not exceed his proper function, which is neither more nor less than to make patients aware of impulses of which they were previously unaware.

The authoress goes perilously near misstatement in another instance, when she writes of the duration of psychoanalysis in terms of months. It is true that patients may obtain considerable relief from their symptoms within a period of months—in fact many receive considerable relief in weeks or even days—but for radical results rather than mere symptomatic treatment analysis should continue for years, three years being generally regarded as the average.

There is one important omission that should be recorded. The authoress makes no mention whatever of what is technically called "negative transference" (hate for the analyst), despite the fact that this is the most important aspect of every analysis.

With these exceptions the book succeeds admirably in its object; and the manner in which the information is presented is both clear and interesting. The most important point stressed therein is the qualification of a properly trained psychoanalyst, including as it does insistence on the necessity of his undergoing a personal analysis as a preliminary to undertaking the treatment of others.

[1] "Psychoanalysis Explained", by D. R. Blitzsten, with an introduction by A. A. Brill, M.D.; 1937. London: George Allen and Unwin. Crown 8vo, pp. 76. Price: 3s. 6d. net.

THE RUXTON CASE.

"MEDICO-LEGAL ASPECTS OF THE RUXTON CASE",[2] by John Glaister, Professor of Forensic Medicine, University of Glasgow, and James Couper Brash, Professor of Anatomy, University of Edinburgh, deals, as set out in the preface, with the medical, legal and other scientific aspects of a case without parallel in criminal records, namely, the murder and mutilation by Dr. Ruxton of his wife and maid. It is claimed also that the history of the case emphasizes the high importance of "team work" by medical experts. At first sight there seems to be an almost excessive number of experts, but apparently they did not get in one another's way.

There is set out in minute detail a description of the dismembered bodies, the identification of the bodies, and much other interesting matter. The estimation of the stature of body number 1 was calculated from Pearson's formulæ. In our experience this usually provides a figure one to two inches too small, and as regards the femur, we have found that the old figure given in Quain, that that bone is 0·275 the height, is as a rule more nearly

[2] "Medico-Legal Aspects of the Ruxton Case", by J. Glaister, M.D., D.Sc., and J. C. Brash, M.A., M.D., F.R.C.S. (Edinburgh); 1937. Edinburgh: E. and S. Livingstone. Imperial 8vo, pp. 300, with 172 illustrations. Price: 21s. net.

accurate, as apparently it would have been in this case. Such estimates can be only approximate.

One of the most interesting chapters deals with the skulls and the portraits of the two deceased women, where a description is given of a novel method of comparing the outlines of a skull with the outlines of a life size, or supposedly life size, portrait of the deceased, the skull being finally photographically superimposed on the portrait. The main value of this method would be to prove a negative, and there would seem to be a risk of unfairness to the accused. Some lawyers fight shy of blood grouping in cases of alleged paternity, for fear that the defendant may fall within the possible group, and that this fact may be accepted as positive evidence against him. This objection could be raised perhaps even more strongly against the photograph. Still, that is a matter for the defence to watch. In any case the evidence, apart from this, was more than sufficient to convict Dr. Buck Ruxton. One has no difficulty in agreeing with the learned judge when he stated that never had he seen expert witnesses more careful and more eager not to strain a point against an accused person.

An interesting feature was the presence amongst the remains of a Cyclops eye, though, as stated in the text, there were two eyes so close together without any intervening bone that they had been recognized as the eyes of a Cyclopean monster. The presence in the chorioid of a tapetum showed that it belonged to an ungulate animal, but not a pig. Probably it was as suggested, a preserved anatomical specimen.

The working out of the Ruxton case reflects the greatest credit upon all the scientists engaged, as well as upon the Scottish and English police, to whom the book is dedicated. The book is beautifully produced, and the description is so clear that it is a pleasure to read it. Everyone interested in medico-legal work should study it.

MEDICAL BACTERIOLOGY.

IN his preface Dr. Fairbrother states that his book, "A Text-Book of Medical Bacteriology", is an attempt to deal with "bacteria as agents of disease in man, and with the application of bacteriological methods in the prevention, diagnosis and treatment of disease".[1] The attempt has been unusually successful. Throughout the book the medical application of bacteriology has been stressed, particularly in those sections which deal with infection and immunity, and attention is directed to the fact that the study of bacterial metabolism and of the mechanism of the various immunological reactions has become so complicated that the assistance of both chemists and physicists is required in attempts to solve the various complicated problems.

The nomenclature recommended by the Society of American Bacteriologists has been followed in the text, which is divided into three parts. Part I deals with general bacteriology. It includes a short but excellent historical survey—an aspect of bacteriology too frequently overlooked—and a helpful chapter on the application of bacteriology to medicine. Part II is devoted to systematic bacteriology, and in connexion with each group of organisms reference is made to recent developments. Thus in the chapter on the staphylococci and toxin production the work of Burnet receives attention and the use of staphylococcal toxoid is discussed. In the section on streptococci, while a tribute is paid to the assistance afforded by the precipitin tests of Lancefield and the agglutination of the various strains of hæmolytic streptococci, it is recognized that a relatively simple method of differentiating these strains is not yet available to the clinical bacteriologist. A description of the normal antigenic structure of members of the Salmonella group makes clear the necessity for the use of at least six suspensions in routine agglutination tests when infection due to a member of this group is suspected. The chapter on the filtrable viruses is presented with the authority and restraint of one who has engaged in original work in this field. In Table XXXI, which divides the virus diseases of man into (i) diseases of accepted virus origin, (ii) diseases of probable virus origin, and (iii) diseases of possible virus origin, the wide field still unexplored in the study of this group of organisms is convincingly presented. Part III is devoted to general technique and includes short chapters on the microscope and staining methods, on the preparation of culture media, and on serological technique.

This book will prove useful and interesting to all those engaged in the study of bacteria, and the simple style is particularly effective in dealing with some of the more complicated aspects of bacteriology.

Of relative unimportance we note: (i) that in the use of alum-precipitated toxoid in diphtheria immunization the second dose recently recommended by most authorities was not considered important, and (ii) that decolorizing with alcohol as well as acid was referred to as a "particularly useful" and not as an essential step in staining urine and fæces by the Ziehl-Neelsen method.

[1] "A Text-Book of Medical Bacteriology", by R. W. Fairbrother, D.Sc., M.D., M.R.C.P.; 1937. London: William Heinemann (Medical Books) Limited. Medium 8vo, pp. 445, with illustrations. Price: 15s. net.

Notes on Books, Current Journals and New Appliances.

CLINICAL REVIEWS FROM PITTSBURGH.

FROM the house of Paul B. Hoeber, of New York, and under the editorship of Dr. H. M. Margolis, comes a book entitled "Clinical Reviews of the Pittsburgh Diagnostic Clinic".[1] Its subtitle is "Guideposts to Medical Diagnosis and Treatment". There are eight contributors. The subjects are treated in essay form and include: "Constitutional Biologic Inadequacy", "The Psychoneuroses", "Anorexia Nervosa", "Attacks of Unconsciousness and Convulsive Seizures", "Hyperthyroidism", "Hypothyroidism", "Diabetes Mellitus", "Obesity", "Coronary Disease and Angina Pectoris", "The Treatment of Renal Disease", "Pernicious Anæmia", "Indigestion", "Some Causes of Rectal Bleeding", "Jaundice", "Peptic Ulcer", and so on. One of the most useful features of the book is that each chapter concludes with some suggested bibliographic references.

UROLOGY FOR THE GENERAL PRACTITIONER.

THE object of Dr. Koll's treatise[2] is an excellent one and the book should be of value to the general practitioner. The volume is a small one to deal with such a large subject, and the author has done well to cover the ground as fully as he has done.

More space might have been devoted to descriptions of abnormal physiology, such as is involved in prostatic obstruction, in severe urethral stricture, and in renal ptosis; and perhaps less to special lines of treatment which are really of a urological nature. Otherwise the book should be of value.

[1] "Clinical Reviews of the Pittsburgh Diagnostic Clinic: Guideposts to Medical Diagnosis and Treatment", edited by H. M. Margolis, B.S., M.D.; F.A.C.P.; 1937. New York: Paul B. Hoeber Incorporated; Australia: Angus and Robertson Limited. Medium 8vo, pp. 573. Price: 32s. 6d. net.

[2] "Medical Urology", by I. S. Koll, B.S., M.D., F.A.C.S.; 1937. St. Louis: The C. V. Mosby Company; Australia: W. Ramsay (Surgical) Proprietary Limited. Medium 8vo, pp. 431, with 92 text illustrations and 6 colour plates. Price: 30s. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 25, 1937.

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THE STUDY OF THE HISTORY OF MEDICINE IN AUSTRALIA.

STUDY of the history of medicine throughout the Commonwealth of Australia can scarcely be termed general. Certainly, there are, here and there, individual medical practitioners who have made the history of the healing art, in the world at large or in certain parts of it, their own particular and absorbing hobby. The practical sequel to interest of this kind has always been the collection and study of old medical books, papers, journals and instruments, as far as individual finances and buying facilities have allowed. Such collections are willingly displayed to the interested visitor, or to an audience at a medical meeting, or even, as latterly, at the hobbies exhibition of a medical congress. When the time comes, as it eventually must, for the disposal of a collector's worldly treasures, these playthings upon which he has bestowed much attention and affection are too often dissipated. By devious ways some may reach the appreciative hands of another devotee, and so at times again be available to the medical public. Many other literary relics, their value apparently lessened by their fringed edges and broken backs, suffer many obscure indignities, even total or partial incineration.

The American is often considered as the apotheosis of modernity. He is always completely up to date in everything. He is even held to be rather scornful of older institutions and culture. His busy existence would seem to leave no opportunity for poring over old books or even spending much time in a sympathetic consideration of the past. Yet the American is very much interested in medical history. Nearly all the principal medical schools support a chair of medical history. Some of the rarest and most precious medical manuscripts have found an honoured resting place among these very modern people. Each American graduate has implanted in him the beginnings of a life-long reverence for and interest in the fathers of medicine, their writings, precepts and observations. Their historical museums and collections are, moreover, made available to the visitor in a most interesting and stimulating spirit. A courteous curator may even permit the handling and perusal of such treasures by an approved student. A specially trained staff will extract, copy or photograph some pertinent matter which may be of interest or value elsewhere. Exchanges of duplicate copies and loans for special exhibitions are routine practices, to the common advantage of museums or collectors. Catalogues are continually under revision, so that all who care may determine exactly what additions have been made to these warehouses of history. In this country, even Australian history is forgotten or neglected by the great majority of our countrymen. The importance of medical history was unrecognized by the universities until quite recently: a lectureship now exists at the University of Sydney. The sections of medical history within the British Medical Association depend for their existence upon the enthusiasm and personal efforts of half a dozen members.

The time is ripe for a general awakening of interest on the part of the Australian doctor in the story of medical progress in his own and other

lands to which he often seems to be an insufficiently grateful heir. Interest is best aroused, first, by a continuance of regularly contributed articles of historical interest in the medical Press. During the past three years our readers have been very fortunate in this regard. Secondly, the Royal Australasian College of Surgeons and the Australasian College of Physicians could well emulate their parent organizations by the practical encouragement of this branch of knowledge. This interest may be aroused and perpetuated by appropriate historical museums or collections housed in the college buildings themselves. Collectors should be invited to bequeath some or all of their material to the perpetual care of such an institution. College lectures in the history of medicine could perhaps be endowed and so become an annual inspiration. Similarly, university lectureships should be established in Melbourne and Adelaide and ultimately in the University of Queensland; these lectureships may some day become permanent chairs wherefrom may be implanted in students the germs of a lasting interest in the long line of thinkers, pioneers and courageous men who have contributed to the relief of human suffering. There are in our midst today men who are well fitted to tell us more of these things.

Current Comment.

LOBELINE AND THE TOBACCO HABIT.

THERE are certain physical conditions in which the use of tobacco by smoking is prejudicial. Amongst those may be included gastric and duodenal ulcer and circulatory impairment involving the peripheral circulation, as in *thromboangiitis obliterans* and other states in which embarrassment of the circulation is a menace to the patient's health or actually endangers his life. Few persons can voluntarily relinquish the habit of smoking. With some deprivation may be a discomfort; with others it may entail profound misery. To overcome the habit replacement of smoking by mastication of gentian or quassia root, or, in the United States of America, chewing gum has been tried without conspicuous success. Application of a solution of silver nitrate to the buccal mucosa has had some few successes. No special method, however, has been universally satisfactory. In 1936 J. L. Dorsey reported favourably on the use of lobeline sulphate in controlling the tobacco habit. Lobeline is the main alkaloid of *Lobelia inflata*. This plant is known as "Indian tobacco", although there is no convincing evidence that Indians ever used it as tobacco is employed.

I. S. Wright and D. Littauer have made an experimental study of the use of lobeline in tobacco smoking.[1] They inform us that lobeline was available only in an amorphous form until Hermann Wieland extracted a crystalline compound in 1915. The formula of the pure alkaloid was determined by Heinrich Wieland in 1921. Two commercial preparations of so-called lobeline sulphate in use are Merck's preparation, which appears to be a mixture, in unknown proportions, of the sulphates of α, β and γ lobeline, and Mallinckrodt's product. This seems to be a mixture of the sulphates of all the lobelia alkaloids. There is also a crystalline hydrochloride of α lobeline, which is sold under the trade name of "Alpha-Lobelin". Wright and Littauer used capsules containing 0·008 gramme of lobeline sulphate with an inert base of magnesium oxide or starch. The preparations of Merck and Mallinckrodt were employed in approximately equal numbers. In treating the tobacco habit it is rightly mentioned that the usefulness of any "cure" will be greatly restricted if it be too disagreeable. Wright and Littauer concluded that the effects of ingestion of lobeline were too unpleasant to warrant its use for the tobacco habit in the doses recommended. The unfavourable manifestations of its exhibition included eructations, nausea, epigastric discomfort, vomiting and loss of appetite. It is insisted that patients will not willingly take a drug that causes such symptoms at unpredicted times. The crystalline pure salts were just as bad in this respect as the amorphous drug. The experiments, although not conclusive, indicated a trend towards a nicotine-like action on the circulation of blood through the small vessels of the extremities. Also, as with nicotine, in some instances following ingestion of lobeline there was a drop in skin temperatures. In none was there an upward tendency. W. J. R. Camp, in 1927, showed that, when the superior cervical ganglion of a rabbit was painted with α lobeline, a constriction of the ear vessels was seen, followed by dilatation. Stimulation of the ganglion or preganglionic fibres produced no effect. Constriction, however, followed stimulation of post-ganglionic fibres. This demonstrated that the ganglion was no longer able to transmit impulses. Camp concluded that, as with nicotine, the change in the calibre of the blood vessels and, accordingly, the change in blood pressure, were mostly due to stimulation of the ganglion cells to the blood vessels. A rise of blood pressure was due to activation of the constrictors and a fall to activation of the dilators and vagus action on the heart. Wright and Littauer emphasize the fact that any drug which causes vaso-constriction for even a brief period is contraindicated in circulatory disease due to spasm or occlusion of blood vessels. In the present series,

[1] *The Journal of the American Medical Association*, August 28, 1937.

when lobeline was given by mouth, any effect on the blood sugar level was not constant, although considerable rises were noticed in two cases. When tobacco smoke was inhaled there was either no change or a rise in the blood sugar level occurred. There was no demonstrable change in the respiration in those who took varying amounts of lobeline over a period of days or in those who took single doses of eight milligrammes (one-eighth of a grain), except in such subjects as had nausea or epigastric pain. The blood pressures of several members of the former group also evinced no alteration.

The question of dosage of lobeline is of great importance. It is possible that a lesser quantity of the preparation may have the required result without producing the unpleasant results. Wright and Littauer tried four patients with a dosage of four milligrammes (one-sixteenth of a grain). One stated that his cigarette smoking had been completely abandoned, with improvement of appetite and a feeling of general well-being. One suffered from mild nausea which prevented smoking. One had an unsettled feeling, but there was no effect on his desire for smoking. The other patient felt no effects of any sort. The undesirable effects of the drug make it self-limiting as regards dosage, but Wright and Littauer issue a warning against the large doses mentioned by Dorsey—particularly in such patients as suffer from cardiac disorder or peptic ulcer.

Lastly, the problem of the permanent status of the patient regarding his former habit must be considered when the habit has been entirely abandoned and substitution medication is unnecessary. Wright and Littauer relate the experience of a patient suffering from *thrombo-angiitis obliterans*. From being a heavy smoker he had reduced his allowance to two or three cigarettes daily. He took one eight-milligramme capsule of lobeline daily for three days, during which he had no wish to smoke. At the same time his appetite depreciated so alarmingly that he discontinued the medication after the third day. On the fourth day he experienced no inclination to smoke, but on the fifth day the urge returned and he smoked a cigarette. It is emphatically pointed out that no substitution product which will tide a patient over a period of transition from being a smoker to a non-smoker, will prevent him from resuming the habit when the drug is discontinued. Wright and Littauer state that only the combination of a clear and emphatic explanation by the medical attendant with determined cooperation on the patient's part will achieve the required result. They do not think that lobeline sulphate administered by mouth in a dose of eight milligrammes is suitable for general use as a "cure" for the tobacco habit. Lobeline itself has a nicotine-like effect on the peripheral circulation. In some subjects vaso-constriction occurs, with a fall in surface temperature. This is not as constant, however, as the effect following the smoking of a cigarette. Like nicotine, lobeline produces in some individuals a rise in blood sugar.

The experiments of Wright and Littauer clearly demonstrate the fact that we have not yet found a satisfactory cure for tobacco smoking. In any case the lobeline method is merely similar to the addition, in nauseant doses, of antimony to alcohol to cure the drink habit.

Special Abstract.

THE MECHANISM OF ACCOMMODATION.

As the result of much laborious work, Edgar F. Fincham has ably elaborated the work of Hess and Gullstrand, who believed that the deformation of the lens which occurs in accommodation could be explained by differences in thickness of the lens capsule at different points, and that since the anterior central part of the capsule was the thinnest, a central anterior lenticonus naturally followed in that situation.[1] The object of Fincham's investigation has been to determine the physical mechanism by which the refractive power of the eye is altered in accommodation. The information concerning fundamental changes in the form of the crystalline lens is now well established, but it is possible to place on these and associated appearances such a variety of interpretations that many antagonistic theories of the mechanism still exist. Since the lens is non-muscular, accommodative changes must be the result of alterations in the forces applied to it by those parts of the eye—the ciliary body, the zonule and the vitreous—which are associated with it; but since these are invisible in the living, normal eye, direct observation is not possible. Rarely, however, opportunities present themselves for observation of the aniridic eye, in which the whole lens, the zonule and the inner edge of the ciliary body are visible; the records obtained from such sources are described in Fincham's monograph. The evidences obtained from the living eye, while not giving a complete solution, strongly point to the explanation of the phenomena. The most conclusive evidence is derived from experiment on the freshly excised young eye, experiment devised to reproduce, artificially, the same conditions as may be concluded on anatomical and physiological grounds to occur during accommodation.

Descartes held, in 1677, that accommodative change was brought about by the fibres responsible for the suspension of the lens, though he was unable to explain the methods by which the fibres operated. Though this hypothesis was correct, it was unsupported by experimental evidence and was therefore not accepted. The theory of an increase in the axial length of the eyeball was espoused by Sturm and many others up to as late a date as 1800. It remained for the genius of Thomas Young to coordinate a mass of hypothetical evidence and to advance the theory that the lens, and only the lens, was the site of those changes which permitted the focusing of objects, near or distant. At the same time, Young, unaware as he was of the existence of the ciliary muscle, seems to have thought that the lens itself contained muscular elements. The explanation which finally excluded all other views was that of Helmholtz; he conceived that the lens, during periods of rest, was in a state of constant tension, and, despite much opposition, his views have more than held their ground, though the estimation of the relative value of different factors has been in dispute. It is due, however, to the labours of Tscherning and Pflugk, the results of which were announced in 1909, that the increase in thickness of the lens as due to the forward movement of the anterior surface in accommodation is now accepted. This supported Pflugk's statement that the vitreous had definite form and was not merely a fluid. He believed that during contraction of the ciliary muscle, tension was exerted on the chorioid, the vitreous

[1] "The British Journal of Ophthalmology. Monograph Supplement No. VIII: The Mechanism of Accommodation", by E. F. Fincham, F.Inst.P.; 1937. London: George Pulman and Sons Limited. Royal 8vo, pp. 80, with illustrations.

body thus being compressed against the lens. Because of the peculiar shape of the vitreous, and because of the fact that the anterior part of the chorioid, when under tension from the muscle, will exert upon it a centripetal compression, the vitreous is pressed against the periphery of the posterior surface of the lens, not upon its centre. The anterior surface of the lens is held at its periphery by sustained tension from the zonule. These forces, it was believed, would result in a forward bulging of the anterior surface, the only free region. So were explained the conoidal form of the anterior surface and the formation of a concave zone near the edge of the posterior surface which had been noted by Pflugk and Maklakoff. The former also alleges that the peripheral parts of the lens are prevented from moving forwards, when the lens is pressed on by the vitreous, by the tension of the zonule, assisted materially by the presence of the iris and of the anterior chamber. It may be doubted, however, whether all these can be important factors in the mechanism, as accommodation seems to be normal in cases of aniridia, while the tension of the anterior chamber is reduced during accommodation. The hydraulic theory, advanced in 1920 by Johnson, Noiszewski and Leonard Hill, claimed that the contracting ciliary muscle puts pressure upon the aqueous, confined in the posterior chamber by the iris, as the pupil contracts on the surface of the lens; a compression of the equatorial parts of the lens results, and the anterior surface bulges. But this mechanism requires that the posterior chamber should be watertight, otherwise the loss of pressure would need a constantly increasing contractile effort on the part of the ciliary muscle for accommodation to be uniform. The posterior surface of the iris, crenated in form, seems to make a water-tight junction with the lens impossible, and the fact that accommodation is unimpaired by iridectomy or by aniridia would appear to invalidate this theory. The chief point of disagreement between the two most acceptable theories seems to centre about the action of the ciliary muscle and the forces applied by it to the lens through the zonule; either the muscle relaxes the tension on which the suspension of the lens depends (Helmholtz), or else it increases, or at least maintains that tension. Fincham's object is, firstly, to decide this question, and then, by examination of the apparatus and its behaviour during accommodative processes in the living eye, to determine the complete mechanism.

It becomes necessary to consider the reduction of the amplitude of accommodation with age and its explanation in relation to the theory of this mechanism. The recession of the "near point" with age is commonly believed to be due to changes in the accommodative mechanism of the eye, and not to reduction in the innervation for near vision. There is a divergence of opinion regarding the parts of the apparatus responsible for the deficiency, but the usually accepted opinion is that the power of the muscle is unimpaired until after middle age, while the lens substance, owing to sclerosis, loses the ability to change to the accommodated form. Those holding this view consider that a change in refraction of one dioptre calls for the same amount of ciliary contraction in both young and old. This unit of ciliary muscle contraction (the "myodioptre") is thought to be of constant value, and the power of the muscle in myodioptres is said to exceed the accommodative ability of even the young lens and to be unaffected by age. Thus a presbyope with only one or two dioptres of accommodation employs only a small portion of his ciliary muscle power in reaching his near point, the rest of this power remaining latent; but the young person with higher amplitude makes use of a greater proportion of his total ciliary power. All this is the normal outcome of the Helmholtz theory. If the function of the lens in accommodation is merely to return passively to its normal convexity when the tension of the contracting ciliary muscle is released, it would seem that the degree of freedom which permits the young lens to accommodate one dioptre would produce an equal change in the older, harder lens. The relation of ciliary contraction to deformation of the lens would be equal in young or old up to the ability of the lens to respond, and further ciliary contraction would produce no result except a slackening of the zonule. It is known, however, that presbyopic subjects, whatever their age, cannot maintain a full amplitude of accommodation for long periods without discomfort; positive lenses are necessary for near work although the near point may not be beyond the normal reading distance. At any age effort is needed to accommodate for the near point, and this effort in a presbyope with a dioptre or two of accommodation is probably as great as it is in a child with a high amplitude.

Anatomically, the crystalline lens is biconvex in form, and its two surfaces meet at the equator in a rounded edge. The area of the posterior surface is larger than that of the anterior, so that more of the lens-substance lies behind the equator than in front of it. The exact determination of the figure of the surfaces of the living lens is extremely difficult, and hard to define in geometrical terms. The radius of curvature is probably in the region of eleven millimetres in the central area in the unaccommodated state. The posterior surface of the lens departs somewhat from the spherical form, the radius of curvature in its central region being between 4·6 and 7·5 millimetres. The lens-capsule is a thin, transparent, highly elastic envelope composed of two layers, which are apparently structureless. The lens-substance is composed of fibres lying beneath a superficial layer of epithelium, so increasing in thickness towards the equator as to take the form of concave meniscus plates. By the superposition of new layers of fibres springing from the epithelial cells at the equator, new fibres grow out towards the pole on both surfaces, and owing to the complete enclosure by the capsule, the older elements are not discarded but become more and more compressed towards the centre, while the size of the lens increases throughout life. The increasing hardness of the lens towards its centre leads to an increase in the refractive index in that situation. The variations in this index are unknown, but they are probably irregular and may account for the characteristic star figure appearance of small sources of light. By early middle age there is a discontinuity in the index of refraction of the lens-substance.

The ciliary muscle, essentially part of the uvea, occupies the major part of the ciliary body, and consists of unstriped muscle cells springing from the chorioid as far back as the equator of the eyeball. The muscle forms a prismatic ring round the eye, with the base of the prism at its anterior innner circumference close to the root of the iris. The anatomy of this muscle points to the conclusion that its inner portion acts as a form of sphincter muscle, so that in contraction the ciliary corona from which the lens is suspended by the zonule will be reduced in diameter.

The lens is connected to the walls of the eyeball and to the ciliary body by the zonule and the vitreous humour. The zonule is in reality a variation of the vitreous, and is so-called "gel" formation. A similar condensation of this gel forms the suspensory system of the lens. The zonule may be considered as an elastic membrane, therefore, and as part of the vitreous, rather than as the system of inextensible fibres which it appears to be in fixed preparations. Fincham has succeeded in showing by experiment that the zonule forms an elastic suspension for the lens, using in this work the lens and zonule dissected out from the fresh eye of a sheep. By this means he was able to make graphic records by means of light reflected from a mercury globule on the anterior surface of the lens. There are two methods available for the study of the changes in the living lens during accommodation. The first of these is by measurement of the catoptric images, the second by direct observation of the luminous section of the lens produced by the slit-lamp. In cases of aniridia in which the whole of the lens is exposed to view, the slit-lamp affords a means of watching the changes in accommodation, although direct measurement is difficult. Owing to the laminated structure of the lens and the increasing refractive index of its successive layers, images reflected from its front surface are apt to be badly defined, while there is an overlapping of images from the surfaces immediately behind. But at a suitable angle of observation, the central and brightest part of the image assumes a granular appearance, and this is probably produced by the epithelium which covers the surface immediately behind the capsule. The quality of the images from the anterior surface of the lens varies greatly in different eyes, even if the age and refractive conditions are equal and the eyes healthy. Fincham has noted a few cases in which the images were so bright that

it has been possible to make photographic records of them. There is no doubt that the lens is displaced in the direction of gravity during great efforts of accommodation, but its position is unaffected by gravity in the unaccommodated state. In the case of an eye in which the lens capsule is empty, the difference between the tautness of the capsule in the unaccommodated state and its slackness during an effort of accommodation can be accounted for only by the relaxation of the tension under which the capsule is held. In the dissected eye of a child in which the lens was in the unaccommodated form after the removal of the cornea and iris, the anterior surface of the lens assumed a form consistent with an accommodation of fourteen dioptres after the suspensions of the lens were severed. This evidence appears to confirm the general validity of Helmholtz's theory, but there are still some points which are unexplained by that theory. The conoidal form assumed by the anterior surface of the lens in accommodation is an important factor; it permits a great increase in curvature of that part of the surface exposed by the pupil, without necessitating a large distortion of the whole body of the lens-substance; but the phenomenon of this characteristic surface form remains unexplained by the Helmholtz theory. A further objection to that hypothesis is that it assumes the living lens-substance to be held in a constrained form when the eye is at rest, the lens assuming its free natural form during accommodation only. The loss of accommodation with age also remains unexplained. If, as Helmholtz supposed, the free form of the lens-substance is that of accommodation, and it assumes this form as a result of its own elasticity, it is strange that as the lens hardens with age it should become set in the flat unaccommodated form. And if, as has been demonstrated by Fincham, the reduction in amplitude is caused by loss of the supposed elasticity of the lens-substance, the near-point of the presbyope could be reached with only a small ciliary effort and maintained in comfort. Further, no existing evidence shows that the necessary elasticity for the lens-substance does in fact exist; in Fincham's experiments on young monkeys it was found that the removed lenses had the characteristic accommodated form, but when the capsules were removed, the lens-substance assumed the unaccommodated figure. This would not have occurred if accommodation had been due to the lens-substance's elasticity. Since it has been proved that the change in lens-form is produced without the agency of external forces, and as the lens-substance does not possess the necessary elasticity to allow of its changing to the accommodated shape when the tension upon it is released, it must be concluded that the force required is provided by the capsule which is possessed of the necessary elastic property. In a study of the relationship between the thickness of the capsule and the form of the lens, Fincham has demonstrated that in the primates, in which the amplitude of accommodation is high, the capsule covering the anterior surface is relatively thin at the centre, where the accommodated surface is steeply curved, and that a zone of greater thickness surrounds this area and corresponds to that part of the surface which is relatively flatter. In the eyes of lower mammals in which the lens surface does not take up the conoidal form in accommodation, the capsule is uniform in thickness over the anterior surface. It therefore seems that the accommodated form of the lens is determined by the nature of its capsule. As a general statement of the mechanism of accommodation, it may be said that when the eye is in its passive state with the ciliary muscle at rest, the lens capsule is held under tension by the elastic zonule and vitreous, by which it is suspended from the wall of the eyeball, and the zonule is then stretched. The lens substance is then in its normal undistorted form, and the capsule exerts no influence upon it. The muscle contracting, the ciliary ring is reduced in diameter and the tension of the lens suspensions is lessened. The zonule is not now so fully stretched, and the elastic capsule, under the freedom now permitted it, presses upon the soft lens-substance, moulding it into the accommodated form. This results from pressure at the equator and in those regions where the capsule is thickest, while there is bulging in the thinner parts. It will be seen, then, that normally the crystalline lens is in the passive state; the accommodated form is impressed upon it by the capsule when it is freed to do so by the contraction of the ciliary muscle. The only structures under tension when accommodation is at rest are those which are "non-living"—the capsule, the zonule and the anterior part of the vitreous.

There is a demonstrable relative immobility of the posterior surface of the lens. One cause of this is that owing to the great thinness and hence flexibility of the posterior capsule it is probably fully distended when the suspensions are under tension and accommodation is not being carried out. It has been shown in the case of the empty lens-capsule that this posterior membrane, unlike the anterior, does not assume a definite form when an effort of accommodation is made. It is probably too weak to have an effect upon the form of the lens-substance and is probably at all times fully distended. Again, the posterior surface of the lens is much more curved and is of greater area than the anterior, so that when alterations of tension occur at the circumferences of both these surfaces they have a greater effect upon the smaller area.

The symptoms of presbyopia are to be explained by the function of the lens capsule in accommodation in relation to the increased rigidity of the lens-substance, and also by the possible effect of loss of power in the ciliary muscle of the senile eye. Microscopic sections of the ciliary muscle of such an eye show a greater quantity of interstitial connective tissue among the muscle bundles than are apparent in corresponding structures in the young eye; and it is well known that the unstriped muscles of the body generally become less active in old age. In spite of this, it is unlikely that the muscle should have lost some of its power at the early age at which the near-point commences to recede. But lens-substance, unique by reason of its peculiar epithelial origin and its continued growth within its capsule without shedding of its older cells, must be subject to progressive sclerosis.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Queensland Branch of the British Medical Association was held on October 1, 1937, DR. R. G. QUINN, the President-Elect, in the chair.

Danger Signs in Childhood.

DR. S. F. MCDONALD read a paper entitled "Some Danger Signs in Childhood" (see page 1107).

DR. P. A. EARNSHAW, in opening the discussion, thanked Dr. McDonald for giving him the opportunity to peruse his paper beforehand. He said that Dr. McDonald's observations were obviously those made in his own private practice; and the great majority of the conditions mentioned, with a few exceptions, were such as would occur in anyone's private practice. Dr. Earnshaw remarked that the minor signs and symptoms had been emphasized. As an example of the need for this he cited a case of tuberculous meningitis. The patient was a child less than a year old, and had been seen by a private doctor who had sent him to a children's hospital for observation. The child was sent home, the parents having been told that there was nothing wrong with it. Dr. Earnshaw saw the child the following day and found it obviously ill. It was drowsy, but irritable when approached. It had a slight rise in temperature, some neck stiffness, and the tension of the fontanelle was increased. The father was a returned soldier who received a pension on account of tuberculosis. The diagnosis of tuberculous meningitis was made and this was subsequently verified at autopsy.

Dr. Earnshaw thought that the afebrile nature of plumbism was sometimes more apparent than real. Dr. Breinl had previously drawn attention to the mild fever which was apt to accompany plumbism; the tempera-

ture generally varied between 37·25° and 38° C. (99·0° and 100·5° F.). The last six patients with lead palsy whom Dr. Earnshaw had had under his care all had this mild fever.

Dr. Earnshaw drew attention to another aid in the differential diagnosis between rheumatism and osteomyelitis. It was sometimes very difficult to distinguish between these two conditions. Great assistance might be had from a blood smear. In rheumatism the leucocyte count seldom rose above 20,000 or 22,000, whereas in osteomyelitis it was generally above 25,000. In rheumatism the percentage of polymorphonuclear cells was usually below 80, whereas in osteomyelitis it was generally above 80.

Dr. Earnshaw said he was glad to hear Dr. McDonald's opinion that teething might produce "other things than teeth". Teething was a physiological process as menstruation and pregnancy were; and just as menstruation and pregnancy provided abnormal symptoms and signs, so might teething.

Dr. Earnshaw also mentioned what a distressing condition enuresis might be, especially when it persisted into the "late teens" or "early twenties", and stressed the need for proper medical advice in dealing with this condition. Another cause of enuresis was *spina bifida*; as in hydronephrosis and dilatation of the ureters, enuresis occurred day and night. Generally speaking, pyuria was also present.

DR. G. W. MASON wished to draw attention to another condition which at times was brought under notice by convulsions, namely, pyelitis. The particular child he had in mind, a female, aged two or three years, looked quite well, though the mother reported that urination had been attended by some straining and discomfort. The pyuria had now lasted from seven to eight years, but of late had much improved, and appeared to be cured after treatment with mandelic acid. Another obscure case was that of a boy who complained of abdominal pain after a long bicycle ride, with guarding of the right side of the abdomen. At operation all that could be found was a long, whitish, fibrinous clot in the region of the foramen of Winslow. Later the temperature rose to 39·45° C. (103° F.) and the patient contracted a subphrenic abscess and was operated upon. This was a case in which the possibility of either pneumonia or appendicitis had to be remembered in the diagnosis.

DR. L. G. HILL, commenting on enuresis, remarked that locally very little urological investigation was being done in cases of pyuria in children. This meant the overlooking of a percentage of more or less marked abnormalities. In minor cases one was apt to prescribe potassium citrate *et cetera*, and perhaps allow the patient to drift into morbidity in adult life.

Speaking of children with protruding abdomen and bad posture, Dr. Hill said that there were encountered often vague abdominal pains (more or less severe) and constipation. Usually aperients in plenty had been tried. Many of these conditions settled down with appropriate treatment. He himself had had success with a mixture of bismuth salicylate, bismuth subnitrate, sodium bromide, tincture of belladonna and *Aqua Menthæ Piperitæ* as a supplement to postural correction.

DR. I. G. HOOPER wished to report on the successful treatment of enuresis in a home for boys with which he had been associated. Acting on the advice given in *The British Medical Journal*, the foot of each bed was raised, beds were made firm with board supports, and no fluids were given after 5 p.m. All boys were required to report with a calendar provided for them on which a star indicated dry nights. They had been constant bed-wetters for up to three years. Six out of seven showed immediate improvement, and at the end of two months four had seven dry weeks, one had two wet nights in eight weeks, and one had one or two wet nights per week. The seventh had two or three wet nights weekly, but was improving since his tonsils and adenoids had been removed.

DR. T. H. R. MATHEWSON said that time spent in consideration of the "commonplace" was time well spent. An adult consulted his medical adviser for pain; a child was brought because his parents were worried. The adult could refuse treatment, but the child was led as a lamb to the slaughter. If the temperament of the adult was to be considered, how much more so that of the child? Referring to boredom, Dr. Mathewson said that this condition should be considered in the fretful child when there was nothing obviously wrong. It might be an only child or a neglected child, when it would be best to send it to a kindergarten or a nursery school. Unless it was checked, boredom might lead to outbursts of temper and to destructive tendencies. If the family situation was not recognized, the child might be handicapped for life. In an obstinate child constipation might be a refusal to oblige the parents, or a method of retaliation for past slights, real or fancied. If pallor was present a complete blood count should be done. Pallor was due to a variety of causes, but a lymphocytic leuchæmia might be missed if no blood count was done.

Speaking of pneumonia and the acute abdomen, Dr. Mathewson said that one child, aged ten months, who had been referred to a surgeon, had screamed for an hour. It was given *Pulvis Ipecacuanhæ Compositus* and soon afterwards became flushed, with rapid grunting respiration and a temperature of 40° C. (104° F.). It had a board-like upper abdomen, and, until a few whiffs of chloroform had been given, which relaxed the muscles immediately, it was difficult to come to a decision. The child was well in a few days. The condition was probably diaphragmatic pleurisy. With regard to fever in babies, Dr. Mathewson said that often nothing could be found beyond a mild tonsillitis.

Dr. Mathewson thought that both clinic nurses and mothers were familiar with certain conditions associated with teething; a mother would say that her child suffered from "bronchitis" every time he cut a tooth.

DR. E. CULPIN, referring to teething, said that when he was in general practice he had made use of the minor operation of lancing the gums with evident relief to the small patients.

DR. R. G. QUINN conveyed the thanks of the meeting to Dr. McDonald and, before calling on him to reply, remarked that the type of child which convulsed easily was usually deficient in calcium.

Dr. McDonald, in reply to Dr. Mathewson, said that constipation and anorexia were often a revolt in desperation against the parents. Tonsillitis was often found to be a cause of fever with no further signs of disease. In reply to Dr. Earnshaw, Dr. McDonald said that in tuberculous meningitis there was nothing to be done and one should refrain from buoying up the hopes of the parents.

Referring to the fever of plumbism, Dr. McDonald thought that a most important group were those afebrile cases which lay dormant till a bout of fever, such as diarrhœa, suddenly brought the condition to the surface, and one then observed loss of power in the patient's hands and feet. Another type ran a fever as had been stated.

Dr. McDonald also wished to emphasize the use of the leucocyte count in osteomyelitis, and further remarked on the fortunate position of Queensland in the incidence of tuberculosis.

Speaking of enuresis, Dr. McDonald said that there were two children with *spina bifida* at the Home for Crippled Children, Montrose; they looked well, but were absolutely hopeless. In reply to Dr. Culpin, Dr. McDonald agreed that other authorities had found that gums in teething were eased by lancing. In reply to Dr. Mason, Dr. McDonald said that he had found pyelitis amongst the fevers producing convulsions.

Dr. McDonald congratulated Dr. Hooper on his success in his psychical competition and remarked that the child felt that something definite was being done to assist it. In one case, Dr. McDonald observed, bed-wetting went on to the age of fourteen years, when the child was sent to a boarding school. The condition did not ease till the boy

was eighteen years of age. Two other sufferers went till the age of twenty-five years.

In ptosis, Dr. McDonald said, drugs were of value, but they should be used with general exercises to produce confidence.

A MEETING of the Victorian Branch of the British Medical Association was held at the Alfred Hospital on Wednesday, July 21, 1937. The meeting took the form of a series of clinical demonstrations by members of the staff of the Baker Institute and of the honorary medical staff of the Alfred Hospital.

Asthma and Hay Fever.

DR. CHARLES SUTHERLAND showed six patients to illustrate methods of investigation and treatment for asthma and hay fever. The first patient was a man, aged thirty-eight years, who had had hay fever and slight asthma each summer between November and January for eight years. He had noticed that dust from horses would also cause these symptoms at any time. Skin tests revealed marked sensitiveness to horse dander and also to pollens of capeweed and several grasses. Desensitization with a mixture of these was started in June, 1936, and he was free of symptoms during the summer, except for one attack which lasted for two hours. The form of treatment was being repeated.

Another patient shown by Dr. Sutherland was a girl, aged fifteen years, who had had asthma for two years. The attacks had occurred at all times of the year, but she had noticed that she was much freer from attacks in Melbourne than in Gippsland. Skin tests revealed sensitiveness to horse dander, to feathers, to house dust and to capeweed pollen, and Dr. Sutherland said that an attempt would be made to desensitize her with a mixture of these.

Dr. Sutherland showed another patient to illustrate the beneficial effects of lipiodol applied therapeutically to the bronchial tubes. The patient shown was a woman, aged thirty years, whose asthma had commenced eight years previously, after a series of severe colds. Skin tests revealed no signs of sensitiveness to pollens, dusts *et cetera*, but the von Pirquet test revealed a small positive reaction to both human and bovine tuberculin. The skiagrams of the lungs were of normal appearance, and those of the antra indicated the presence of very moderate thickening in the antral walls. No tubercle bacilli were found in the sputum on repeated examination. Dr. T. Wynne had injected warm lipiodol into the trachea once a week, with the patient under local anæsthesia, and definite improvement had followed. The patient had been able almost entirely to cease injecting adrenaline. She was also having injections of tuberculin as a non-specific treatment, and could at the time of the meeting tolerate 0·4 cubic centimetre of the 1 in 100 dilution.

Dr. Sutherland also showed a woman, aged fifty years, who had had severe hay fever every spring for over fifteen years. Skin tests revealed large reactions to all the grass pollens tested, and also to pollens of sunflower, of dahlia and of cosmos. Desensitization carried out between July and November, 1936, had given a great measure of protection, and treatment was being started again. The patient had suffered from dermatitis of the face each summer, but had had no recurrence of it during the summer following the treatment.

Dr. Sutherland's fifth patient was a girl, aged eighteen years, who had had nasal obstruction, rhinorrhœa, sneezing and slight asthma for over two years. Skin tests revealed sensitiveness to feathers and to house dust, and treatment by desensitization was being started at the time of the meeting. Dr. Sutherland said that it was, of course, essential for these patients to avoid as thoroughly as possible the irritants to which they were sensitive.

The final patient shown by Dr. Sutherland was a woman, aged thirty years, who had had asthma for over six years and who had reacted to horse dander, to cattle hair, to house dust, to kapok, to linseed, to milk and to pollen of sunflowers. She was often worse in the spring and autumn, and had found that she was worse in her home in New South Wales than in Melbourne. Desensitization with a mixture of horse dander, house dust, kapok and linseed had produced some improvement three years prior to the meeting; but only a very short course had been given. The treatment had been restarted in March, 1937, with remarkable improvement in her condition. Dr. Sutherland said that the injections had produced very large local reactions, and it had been necessary to go very slowly with the increases in dosage.

MISS SHARWOOD, B.Sc., showed charts illustrating the variations in pollen content of the atmosphere at different seasons, and also demonstrated methods of performing skin tests for sensitiveness.

Pulmonary Tuberculosis following Silicosis.

DR. P. W. FARMER, JUNIOR, showed a male patient, aged seventy years, who had first presented himself at the hospital four years prior to the meeting because of precordial pain and breathlessness on exertion. The patient had previously been a gold miner for many years. Examination of his chest had shown that his heart was situated well to the right of the sternum, the right border being 6·875 centimetres (two and three-quarter inches) to the right of the sternum; his condition had been erroneously diagnosed as dextrocardia. A radiographic examination of his chest revealed gross fibrotic infiltration throughout the right lung and in the middle zone of the left lung. The heart and mediastinum were drawn over to the right side. The appearances were those of silicosis, but changes at the apices suggested superadded tuberculosis.

Dr. Farmer said that several sputum examinations had failed to reveal any tubercle bacilli. The patient had remained fairly well until April, 1937, when a cough had developed with yellowish sputum, the cough being worse at night; there had also been more dyspnœa. On clinical examination the right border of the heart was seen to be 7·5 centimetres (three inches) to the right of the sternum, and pronounced deviation of the trachea to the right could be demonstrated. Skiagrams of the chest at this time showed that the heart was drawn further to the right, and also that the mediastinum and trachea were further displaced. There was more extensive fibrotic infiltration, and cavity formation was present in the right upper lobe, with infiltration at the right base. The patient's sputum had again been examined on several occasions for tubercle bacilli with negative results, and there was no response to the Wassermann test. He had lost several stone in weight.

Dr. Farmer thought that the points of interest in the case were three. The first was the degree of drawing-over to the right side of the heart and trachea, which could be demonstrated clinically. Before any skiagrams had been taken the patient's condition had been diagnosed as dextrocardia. The second point was that skiagraphic examination revealed tuberculous infiltration in the right lung, but repeated sputum examinations had revealed no tubercle bacilli. The third point of interest was the definite tendency for tuberculosis to follow on silicosis of the lungs.

Chronic Gout with Acute Exacerbations.

Dr. Farmer also showed a male patient, aged forty-nine years. The patient had had his first attack of gout twenty-three years previously, when sudden swelling of the right great toe followed by swelling of the left great toe had occurred. Until the time of the attack the patient had been in the habit of taking four to five glasses of beer daily, but he had not touched alcohol since the attack. Since the first attack the patient found that one or several of his joints would swell and become very painful, usually at an interval of about two months; but he had had a free interval for as long as six months. Nearly all his large joints had been involved at one time or another, the knees, hands, elbows and ankles being most frequently attacked. The patient was at the time of the meeting in a very crippled condition, and his right hand and right ankle were extremely red and swollen. Tophi had been present on numerous occasions.

Dr. Farmer said that the patient's cardio-vascular system had remained in good condition; the systolic blood pressure was 130 and the diastolic blood pressure was 80 millimetres of mercury, and the urine had not been found to contain anything abnormal. The blood urea was 30 milligrammes per 100 cubic centimetres; the blood gave no response to the Wassermann test. No uric acid estimation had been made before an attack. The patient had been placed on a purin-free diet, with a diminution of proteins, but it had not helped him very much. Dr. Farmer thought that tincture of colchicum had certainly eased the pain during the attacks, but it had not prevented them. The patient had taken colchicum regularly for the last ten years. "Atophan" had been given in doses of 0·455 gramme (seven grains), three times a day, but had caused some gastric upset, and at one time had produced a mild degree of jaundice; however, glucose and alkalis had not been given with it. Apparently "Atophan" did not prevent an attack. Dr. Farmer remarked that salicylate therapy had helped the patient on several occasions and the pain had been greatly relieved. The patient had not been able to perform any active work for years, but he had been able to get about on crutches between the attacks.

Post-Encephalitic Parkinsonism.

Dr. Farmer also showed a single woman, aged forty-one years, who had had an attack of severe "influenza" five years earlier, when she had been confined to bed for a fortnight. Two years afterwards she had begun to notice lack of energy and tremor of the hands. When first seen by him five months before the meeting, she showed the features of post-encephalitic Parkinsonism; the face was expressionless, with infrequent blinking, and there was immobility of the facial muscles; the muscles of the arms and legs were hypertonic, this condition being equally distributed to flexors and extensors; the reflexes were present, but of small amplitude, and there was a fine tremor of the hands, which became worse when she was addressed. Her relatives said that they had not noticed any emotional outbursts, such as attacks of laughing and crying. No oculo-gyral spasms had occurred; her speech was monotonous and slurring, and there was no reaction to the Wassermann test.

Dr. Farmer said that the patient appeared to grow worse until two months prior to the meeting, when a definite improvement in her condition had been noticed; and from then onwards the improvement had been maintained. Salivation, which had been a troublesome symptom with her from the outset, had ceased during the daytime, although at night it still worried her occasionally. He remarked that at the time of the meeting more expression could be noticed in her face, and there was no demonstrable tremor of the hands, though a greasy condition of the skin of the face was apparent. Her relatives said that the patient was taking more interest in her affairs and that her general health was better; she was eating and sleeping well. She had been treated with tincture of stramonium since she came under observation, and had reached a dose of 2·64 cubic centimetres (forty minims), three times daily, but often she could take it only twice daily, as blurring of vision resulted. Hyoscine hydrobromide in doses of 0·65 milligramme (one one-hundredth of a grain), three times daily, had been tried, but double vision had resulted, and it had not been given again. Dr. Farmer thought that it would be interesting to see how long the improvement would be maintained in this case, as these patients usually progressed steadily on a downhill course, although in some cases arrest had a tendency to occur. He thought it was at least encouraging to see the decrease in the rigidity and tremor, and her general health was undoubtedly better. No massage had been advised, but at this stage, when there was more improvement, he thought it would be worth commencing.

Aplastic Anæmia with Thrombocytopenic Purpura.

Dr. J. F. CHAMBERS showed a male patient, aged twenty-three years, who, during the month of March, 1937, had suffered from a fairly rapid onset of pallor, of weakness coupled with scattered petechiæ and large purpuric areas on the trunk and limbs, of profuse bleeding from a cut finger, of epistaxis and of oozing of blood from tooth sockets. A blood count made within a month of the onset revealed that the erythrocytes numbered 1,630,000 per cubic millimetre of blood and had an average diameter of 7·6μ; the hæmoglobin value was 28% and the colour index was 0·85%. The leucocytes varied in number from 2,000 to 5,000 per cubic millimetre, of which 0·1% were reticulocytes. The bleeding time was 69 minutes. The patient's serum failed to react to the Wassermann test. A test meal was carried out and the findings were normal. The spleen had not been palpable at any time. The patient had received four transfusions of blood, one pint being given each time; bi-weekly injections of two cubic centimetres of "Anahæmin" or of five cubic centimetres of "Campolon" had been given; and 3·9 grammes (60 grains), three times daily, of *Pulvis Ferri Carbonas Saccharatus* were administered orally.

Apart from two temporary remissions the anæmia had been progressive and was accepted as aplastic in type. Idiopathic *purpura hæmorrhagica* was an improbable alternative diagnosis, as the anæmia was more severe than could be accounted for by the amount of blood lost; the film showed no nucleated red cells or other evidences of disordered attempts at regeneration, such as are found in post-hæmorrhagic states; leucopenia with relative lymphocytosis (from 53% to 78%) had persisted throughout, and the platelet count, during a non-purpuric phase, had remained as low as 40,000 per cubic millimetre.

Though the patient had made a practice while engaged in French polishing of cleansing the hands in benzol, this seemed rather a remote causative factor to consider, because he had discontinued the practice for four years. During the year preceding his illness he had regularly handled powdered lead oxide, but, with the exception of the presence in one film of stippled red cells, no evidence of lead intoxication had been found. Dr. Chambers said that as a less hopeless outlook was adopted nowadays towards the aplastic anæmias, and as variations in type were recognized, he proposed to proceed along similar lines of treatment to those he had described, in the hope that by these means the patient might be tided over the period that would elapse before the marrow underwent regeneration.

Demonstration of the Gynæcological Unit.

DR. ROBERT FOWLER demonstrated a clinical records system, with special reference to cancer follow-up methods. This demonstration will be reported later.

DR. J. M. BUCHANAN and DR. H. G. FURNELL showed patients who had been symptom-free for periods of five years and upwards after treatment for carcinoma of the uterine cervix, either by operation or by radium.

Familial Acholuric Jaundice.

DR. H. LAWRENCE STOKES showed two patients suffering from familial acholuric jaundice. One was a little girl, aged four years, many of whose relatives, on the father's side, had had enlarged spleens removed. Since the age of nine months the child had had many "bilious" attacks, which usually lasted one to three days and occurred every three or four months. With each attack the skin became yellowish and the motions were loose. Between attacks she seemed to be fairly well, and she had not had any other serious illness. On examination she was found to be fairly well nourished, but rather pale. The spleen was enlarged but painless; it moved freely with respiration, and the lower edge extended two fingers' breadth below the costal margin. In other respects the general examination revealed no abnormality. The erythrocytes numbered 4,380,000 and the leucocytes 10,620 per cubic millimetre; the average diameter of the erythrocytes was 6·88μ; some polychromasia, anisocytosis and poikilocytosis were noticed in the blood film. Fragility of the red cells commenced at 0·55% and was complete in 0·45% sodium chloride solution.

The other patient with familial acholuric jaundice shown by Dr. Stokes was a girl, aged seven and a half

years, who at the age of five months was found to have an enlarged spleen. The child's skin had been yellowish from birth; between the ages of two and four and a half years she had had several "turns", during which she vomited several times and the skin became greenish-yellow; each "turn" lasted from two to three days. The patient had had only one "turn" in the twelve months prior to the meeting, and with the exception of these "turns" her general health had been satisfactory apart from frequent colds. The patient's father had been found to have an enlarged spleen at the age of eleven years, and his skin had always been somewhat yellow. He had also suffered from several "turns", the first occurring at the age of sixteen years and the second at twenty-two years, and he had had about five afterwards. During the attack his skin became a deeper yellow and abdominal pain and vomiting occurred. He had refused splenectomy. The child shown was thin and pale; the tonsils were small, but not healthy; the spleen was enlarged, extending two fingers' breadth below the left costal margin, but it was painless and moved freely on respiration. Dr. Stokes commented that the patient had attended the Alfred Hospital only once, and that no blood investigation had yet been made, although the mother had stated that an investigation had been carried out recently elsewhere.

Milroy's Disease.

Another patient shown by Dr. Stokes was a little girl, aged two years and two months, whose feet had been swollen since birth, chiefly on the upper aspects, and the right foot had always been more swollen than the left. Her mother thought that as she became older the swelling subsided a little. The child's general health had been very satisfactory, although her feet appeared to feel cold, and her mother had always kept them well covered. The patient's younger brother had been born with the same condition, and had died at the age of eight months from a different cause. The father was alive and had also had a similar condition at birth; the swelling of the right foot had in his case also been more definite than that of the left foot. Three and a half years before the meeting profuse serous discharge had occurred between some of the toes of his right foot, with temporary relief of signs. His general health had been satisfactory, and as a boy he had taken part in athletics. He had six cousins, male and female, all suffering from the same condition; all the affected relatives were on his father's side.

Pernicious Anæmia Treated with "Anahæmin".

DR. JOHN MCLEAN showed two patients with pernicious anæmia who had been treated by intramuscular injections of "Anahæmin" (B.D.H.). One patient, a man aged sixty years, had been first seen in January, 1937. During the four months previous to that date he had been very weak and tired, and he had lost about 12·7 kilograms (two stone) in weight. He was a pale, thin man, and apart from a smooth, red tongue and loss of vibration sense in both legs, nothing of an abnormal nature was found on examination. Fractional test meals revealed the presence of achlorhydria, and skiagrams of the stomach and duodenum were normal. A blood examination revealed that the hæmoglobin value was 33%; erythrocytes numbered 1,000,000 per cubic millimetre; leucocytes numbered 6,000 per cubic millimetre; and the mean corpuscular volume was 167 cubic microns. In the blood film megalocytes and megaloblasts were seen. The patient had been admitted to the hospital, and on January 25, 1937, he was given an intramuscular injection of three cubic centimetres of "Anahæmin". An immediate subjective improvement followed, and on January 29, 1937, it was ascertained that there was a reticulocytosis of 32%. Four weeks later the patient was given another intramuscular injection of three cubic centimetres of "Anahæmin", and on March 24, 1937, a blood count revealed that the erythrocytes numbered 3,800,000 per cubic millimetre and that the hæmoglobin value was 80%. The patient had received five cubic centimetres of "Campolon" intramuscularly each month afterwards, but the blood count had remained at the same level until, commencing from May 18, 1937, he was given 0·13 gramme (20 grains) of *Ferri et Ammonii Citras* three times daily. Two months later the erythrocytes numbered 4,900,000 per cubic millimetre and the hæmoglobin value was estimated at 90%.

The other patient shown by Dr. McLean was a woman, aged fifty-six years, whose condition, eighteen months before the meeting, had been diagnosed by her private doctor as pernicious anæmia. She had been treated by an intramuscular injection of "Campolon" and by a liver preparation given by mouth. The patient responded to treatment and three months later had been given ten cubic centimetres of *Pernæman forte* intramuscularly, after which she had had a severe reaction. Since that time she had had six "Livrox" tablets per day. Dr. McLean said that the patient had first been seen on July 12, 1937, complaining of great weakness and giddiness, a buzzing sensation in the ears, anorexia and flatulence after meals, and breathlessness on exertion. Her complexion was pale yellow. Examination of the heart, lungs, abdomen and cerebro-spinal fluid revealed no abnormality of note. Urobilin was present in the urine, but no bilirubin. Fractional test meals revealed the presence of achlorhydria. There was a reaction to the Fouchet test. At a blood examination the hæmoglobin value was estimated at 30%, erythrocytes numbered 1,500,000, and leucocytes numbered 2,500 per cubic millimetre. The mean corpuscular volume was 113 cubic microns. In the blood film the erythrocytes showed anisocytosis and poikilocytosis, and occasional macrocytes were seen. On July 14, 1937, the patient received an intramuscular injection of four cubic centimetres of "Anahæmin", after which there had been a definite improvement. The hæmoglobin value and the erythrocyte count steadily increased; there was a reticulocyte crisis of 40% on July 20, and the last blood count, made on July 31, 1937, showed the hæmoglobin value to be 60% and the erythrocytes to number 3,000,000 per cubic millimetre. This was exactly double the figures at the original count, which had been done seventeen days previously. With regard to treatment with "Anahæmin", Dr. McLean cited a private case he had had under observation. The patient had been treated for pernicious anæmia since 1928 and had been through the various stages of liver therapy: first, 0·226 kilogram (eight ounces) of liver a day, then a desiccated liver preparation, then "Ventriculin", followed by "Campolon" injections, and for the nine months prior to the meeting he had had a monthly injection of three cubic centimetres of "Anahæmin". During that time his general condition had been better and his blood count higher than it had ever been before.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Speight, Patrick Howard, M.B., B.S., 1937 (Univ. Sydney), 15 Mandolong Road, Mosman.

Books Received.

HYGIENE FOR NURSES, by J. Guy and G. J. I. Linklater; Fourth Edition; 1937. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 220. Price: 5s. net.

CATECHISM SERIES. ANATOMY: THE UPPER EXTREMITY. PART I, by C. R. Whittaker, F.R.C.S.E., F.R.S.E.; Fifth Edition, revised and enlarged; 1937. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 79. Price: 1s. 6d. net.

A PRACTICAL GUIDE TO MASSAGE, by C. I. Carpenter, with an introduction by D. Kats, Ph.D.; 1937. London: Baillière, Tindall and Cox. Demy 8vo, pp. 142, with illustrations. Price: 5s. net.

ANTENATAL AND POSTNATAL CARE, by F. J. Browne, M.D., D.Sc., F.R.C.S., F.C.O.G.; Second Edition; 1937. London: J. and A. Churchill Limited. Large crown 8vo, pp. 606, with 79 illustrations. Price: 18s. net.

THE CITADEL, by A. J. Cronin; 1937. London: Victor Gollancz Limited; Australia: Angus and Robertson Limited. Large crown 8vo, pp. 446. Price: 10s. 6d. net.

SHORT MANUAL OF REGIONAL ANATOMY, WRITTEN FOR THE MEDICAL STUDENT AS AN AID TO A RAPID REVISION OF THE WHOLE SUBJECT, by J. A. Keen, M.B., F.R.C.S.; 1937. London: Longmans, Green and Company. Demy 8vo, pp. 167, with illustrations.

DEVELOPMENTAL ABNORMALITIES OF THE EYE, by I. Mann, D.Sc., M.B., B.S., F.R.C.S., with a foreword by Sir J. H. Parsons, C.B.E., D.Sc., LL.D., F.R.C.S., F.R.S.; 1937. London: The Cambridge University Press (for the British Journal of Ophthalmology); Australia: S. Jaboor. Royal 8vo, pp. 453, with illustrations, of which many are in colour. Price: 50s. net.

PERSPECTIVES IN BIOCHEMISTRY: THIRTY-ONE ESSAYS PRESENTED TO SIR FREDERICK GOWLAND HOPKINS BY PAST AND PRESENT MEMBERS OF HIS LABORATORY, edited by J. Needham and D. E. Green; 1937. London: The Cambridge University Press; Australia: S. Jaboor. Demy 8vo, pp. 369. Price: 15s. net.

PRACTICAL PHYSIOLOGICAL CHEMISTRY, by P. B. Hawk, M.S., Ph.D., and O. Bergeim, M.S., Ph.D., in collaboration with B. L. Oser, Ph.D., and A. G. Cole, Ph.D.; Eleventh Edition; 1937. Philadelphia: P. Blakiston's Son and Company Incorporated. Medium 8vo, pp. 990, with illustrations. Price: $8.00 net.

THE SINGLE WOMAN AND HER EMOTIONAL PROBLEMS, by L. Hutton, B.A., M.R.C.S., L.R.C.P., with a foreword by D. Forsyth, M.D., F.R.C.P.; Second Edition; 1937. London: Baillière, Tindall and Cox. Foolscap 8vo, pp. 190. Price: 5s. net.

THE IMMUNOLOGICAL REACTIONS OF THE FILTERABLE VIRUSES, by F. M. Burnet, E. V. Keogh and D. Lush, reprinted from The Australian Journal of Experimental Biology and Medical Science, Volume XV, Supplement to Part 2. Australia: The University of Adelaide. Imperial 8vo, pp. 111. Price: 10s. net.

EYESTRAIN AND CONVERGENCE, by N. A. Stutterheim, M.D.; 1937. London: H. K. Lewis and Company Limited. Crown 4to, pp. 100. Price: 7s. 6d. net.

CHILD AT PLAY: OBSERVATIONS, by M. Thorburn, with a foreword by S. Isaacs; 1937. London: George Allen and Unwin Limited. Crown 8vo, pp. 187. Price: 6s. net.

SEX IN RELATION TO SOCIETY, BEING THE FIRST ENGLISH EDITION OF VOLUME VI OF "STUDIES IN THE PSYCHOLOGY OF SEX" ABRIDGED AND REVISED, by Havelock Ellis; 1937. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 544. Price: 12s. 6d. net.

OUT OF MY LIFE AND WORK, by A. Forel, translated by B. Miall; 1937. London: George Allen and Unwin Limited. Demy 8vo, pp. 352. Price: 16s. net.

A PRACTICE OF ORTHOPÆDIC SURGERY, by T. P. McMurray, M.B., M.Ch., F.R.C.S.; 1937. London: Edward Arnold and Company. Demy 8vo, pp. 479, with illustrations. Price: 21s. net.

Diary for the Month.

DEC. 30.—South Australian Branch, B.M.A.: Branch.
JAN. 4.—New South Wales Branch, B.M.A.: Council (Quarterly).
JAN. 6.—South Australian Branch, B.M.A.: Council.
JAN. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 14.—Queensland Branch, B.M.A.: Council.
JAN. 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi to xix.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Resident Medical Officers.

SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Assistant Tutor in Anæsthetics.

SCHOOL OF PUBLIC HEALTH AND TROPICAL MEDICINE, SYDNEY, NEW SOUTH WALES: Diplomas in Tropical Medicine and Public Health.

THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Relieving Psychiatrist, Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135 Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178 North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
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